Chest Imaging

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Sources:

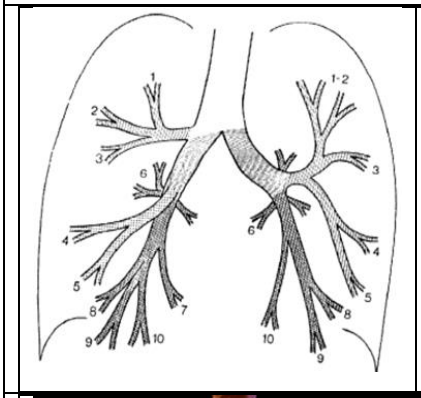
Diagnostic imaging of chest
Primer of diagnostic imaging
Radiopedia
Radiology assistant
Radiographic journal

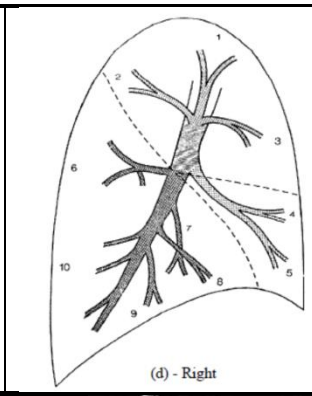
Anatomy

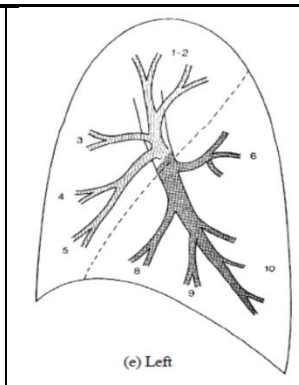
Lobar & Segmental Anatomy

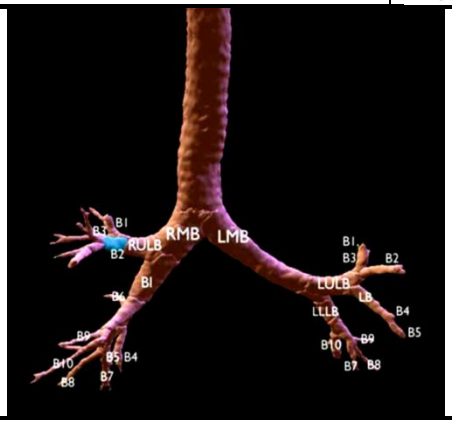
Right Lung	Left Lung
Right Upper lobe: Apical segment (1) Posterior segment (2) Anterior segment (3)	**Left Upper Lobe** Apico-posterior (1+2) Anterior segment (3) Superior lingular segment (4) Inferior lingular segment (5)
Middle Lobe: Medial segment (4) Lateral segment (5)	
Right Lower Lobe: Apical segment (6) Medial basal (7) Anterior basal (8) Lateral basal (9) Posterior basal (10)	**Right Lower Lobe:** Apical segment (6) Medial + Anterior basal (7+8) Lateral basal (9) Posterior basal (10)

Bronchial Anatomy:



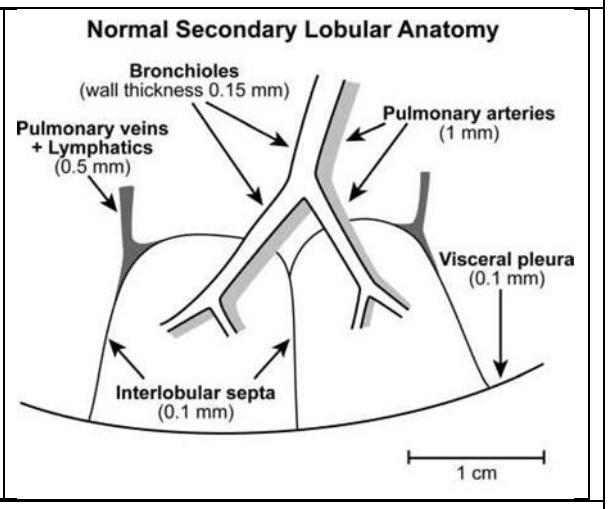






2ry Pulmonary Lobule

- 1ry structural and functional unit of the lung.
- Centre = Centrolobular pulmonary artery + Central bronchiole
- Peripheral = Pulmonary veins + Lymphatics
- Connective tissue = Interlobular septae

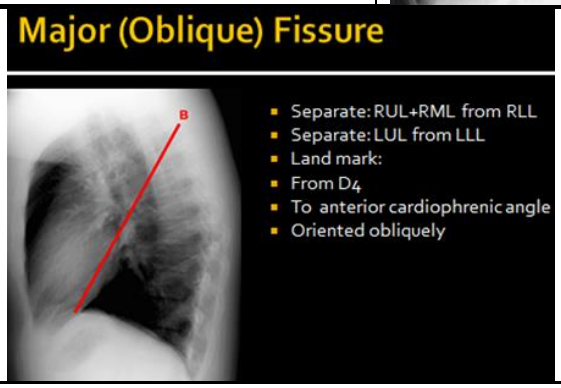


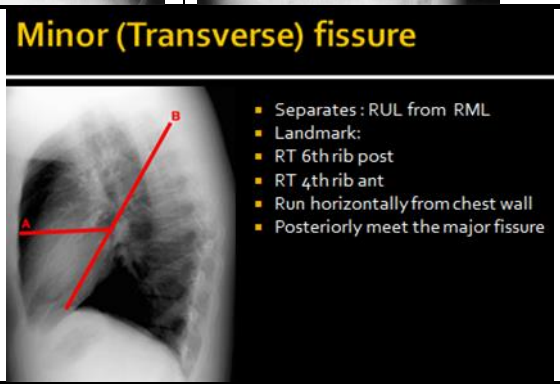
Acinus

Basic unit of gas exchange
containing several generations of branching respiratory bronchioles, alveolar ducts, and alveoli

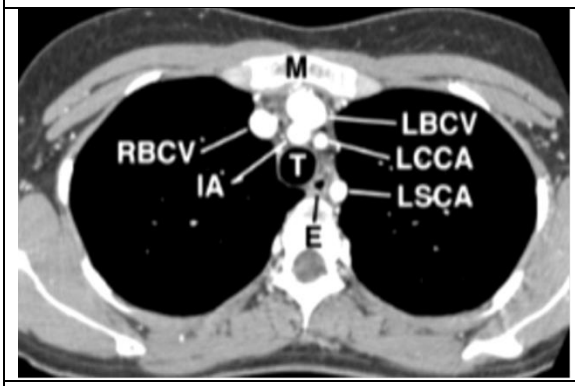
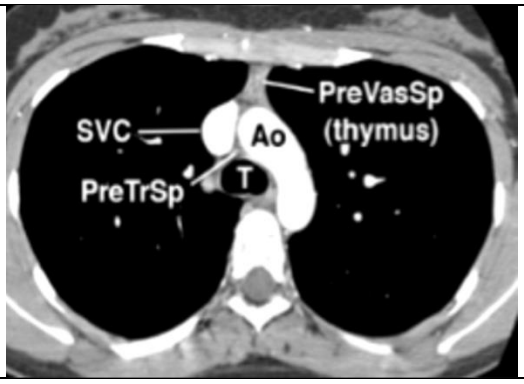
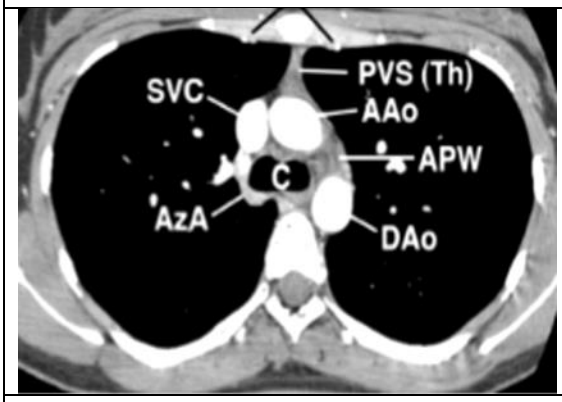
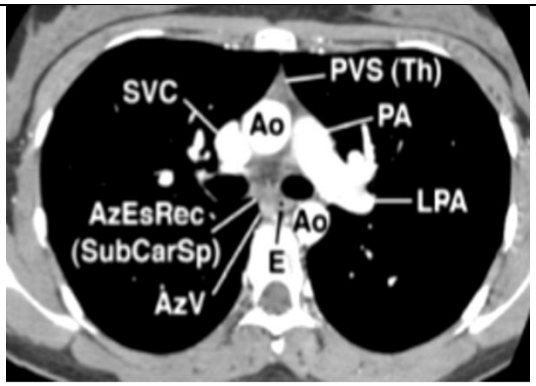
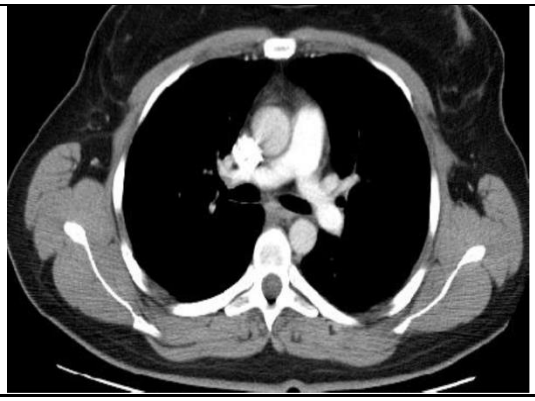
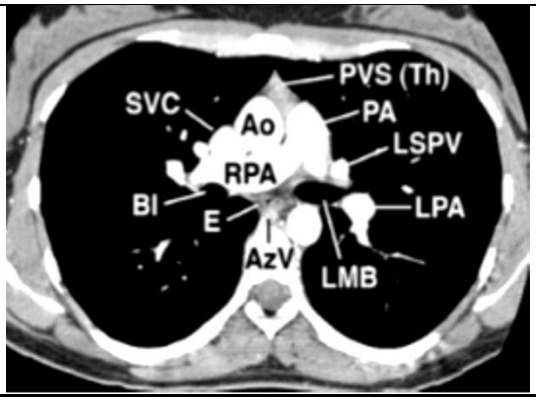
CXR

PA Female	PA Male	Lateral

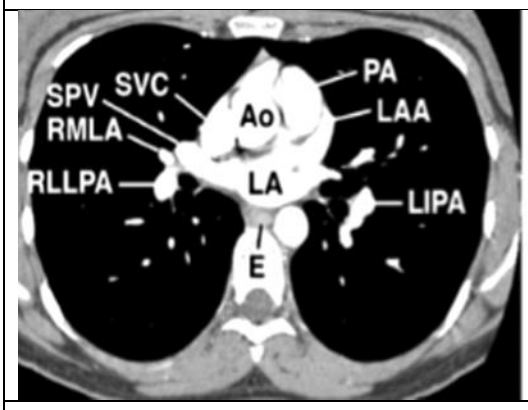


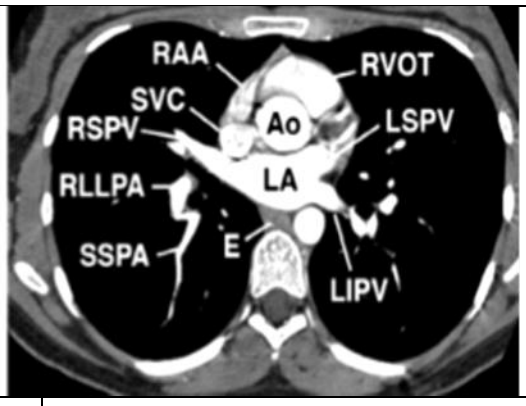


❖ CT Chest Mediastinal Window

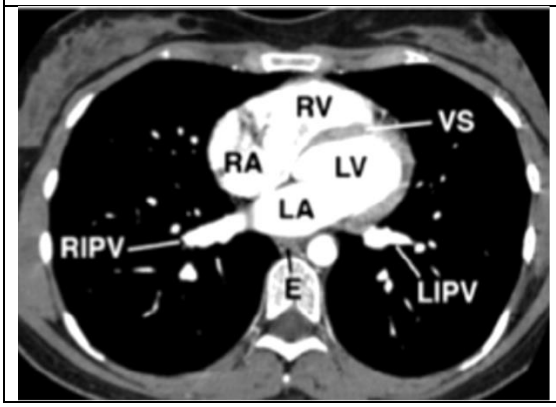
Level above aortic arch	Level of aortic arch
	
Level of aorto-pulmonary window	**Level of LT PA**
	
Level of Main PA + LT & RT PA	**Level of RT PA + LSPV**
	

Level of Ao Root + RVOT + LA

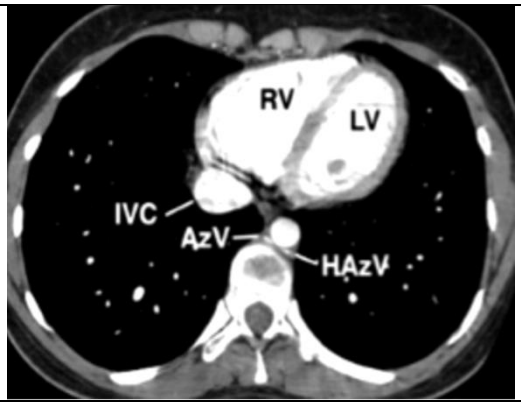




Level of 4 cardiac chambers

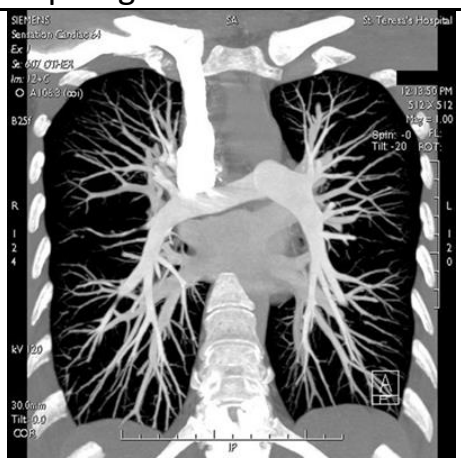


Level of 2 cardiac chambers



❖ CT Pulmonary Angiography

Multislice CT (16 section or more)
Type of contrast: Low osmolar iodinated CM
Amount of contrast = 2-3 ml/kg
Rate of injection = 3-4 ml/sec
Pump injector
Bolus tracking technique.
Slice thickness = 1 or 0.5 mm
Field of view: from the lung apex to the diaphragm



CT Chest Wall 3D Reconstruction

Lung Patterns

Focal / Diffuse

I- Focal Lung Patterns
A) Solitary Pulmonary Nodule **B) Pulmonary Mass** **C) Pulmonary Patch** • **Consolidation** • **Infarction** **D) Pulmonary Cavity**

A- Solitary Pulmonary Nodule

Focal nodular area of increased lung attenuation < 3cm

Differential Diagnosis

Type of Cause	Disease Entity
Neoplastic	
Malignant	Primary pulmonary carcinoma
	Primary pulmonary lymphoma
	Primary pulmonary carcinoid tumor
	Solitary metastasis
Benign	Hamartoma
	Chondroma
Inflammatory	
Infectious	Granuloma (tuberculous, fungal)
	Nocardia infection
	Round pneumonia
	Abscess
Noninfectious	Rheumatoid arthritis*
	Wegener granulomatosis*
Vascular	Arteriovenous malformation
	Infarct
	Hematoma

❖ Benign Versus Malignant Criteria

- Size
- Attenuation
- Margins, contour
- Growth rate
- Nodule enhancement
- Nodule metabolism

Internal characteristics

- Fat
- Calcification
- Cavitation
- Pseudocavitation (Bubbly)
- Air bronchogram
- Reverse halo signs
- Halo sign

1) Nodule Size

- Malignancy positively correlates with nodule diameter.
- The larger the nodule => increase likelihood of malignancy
- The smaller the nodule => the most likely benign.
- Small nodules (<4 mm) < 1% chance of malignancy
- 90% of nodules < 2 cm => benign
- Nodules approaching 3 cm, more likely to be malignant.

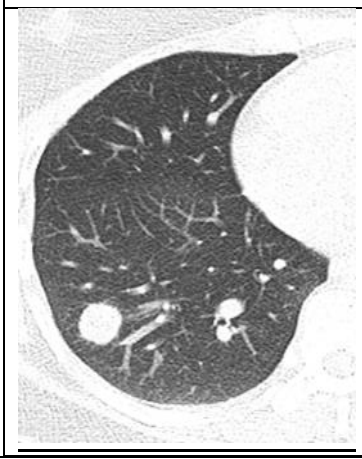
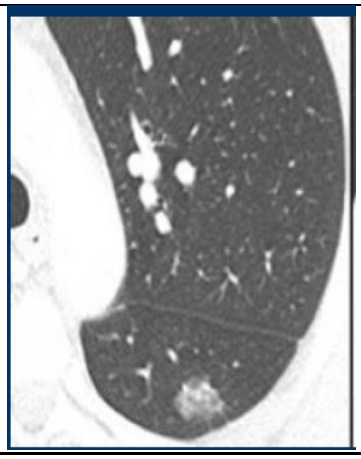
2) Nodule Attenuation:

Subsolid Nodule:

- Ground glass attenuation nodule (GGAN) => 34% are malignant
- Partly solid (Mixed solid and ground glass attenuation) => 50% < 1.5 cm in diameter are malignant

Solid Nodule

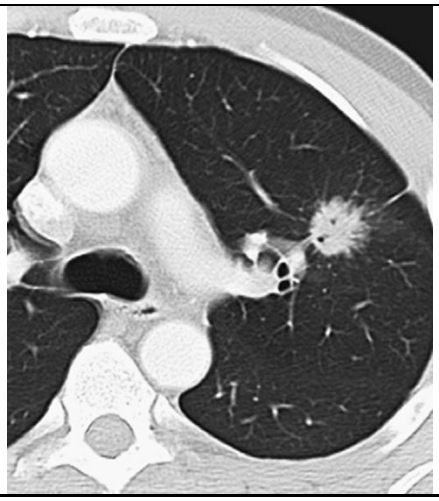
- 7% are malignant; as diameter increases cancer risk increases.

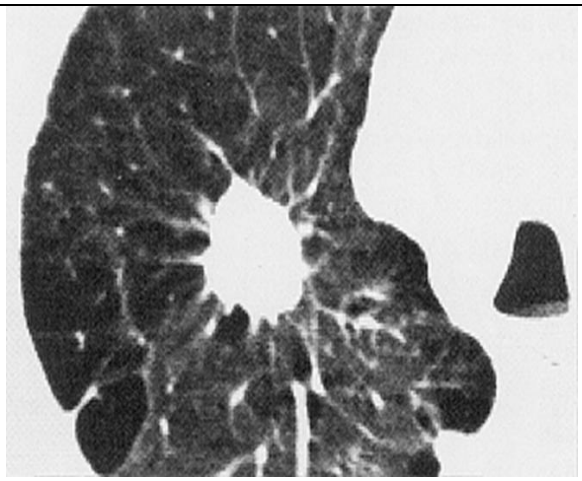
Solid Nodule	Pure GGN	Partly Solid Nodule
		

3) Nodule Margins and Contour:

- Benign nodules
  - ⇨ Well-defined margins
  - ⇨ Smooth contour
- Malignant nodules
  - ⇨ Spiculated margins (sunburst or corona radiata sign)
  - ⇨ Lobular or irregular contour

❖ **Corona radiata = Speculated margins of pulmonary nodule with linear extension to the pleura**

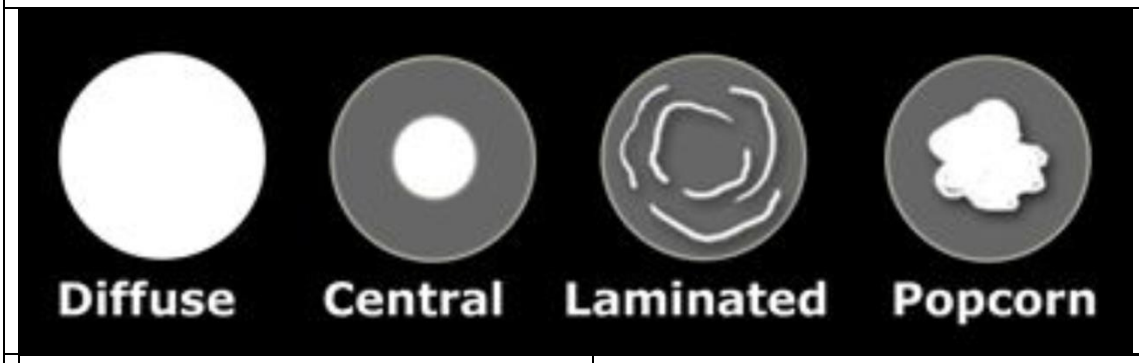




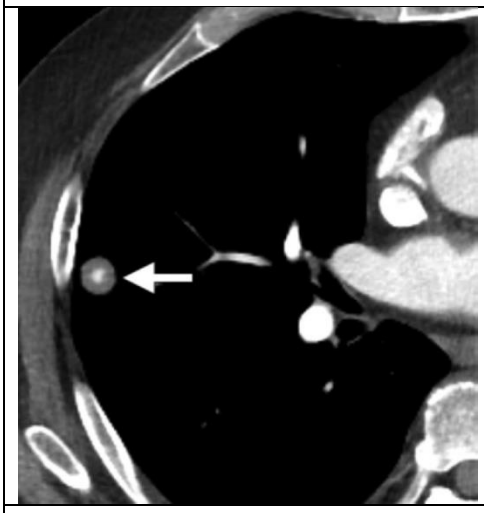
4) Calcification

<u>Benign patterns of calcification:</u>

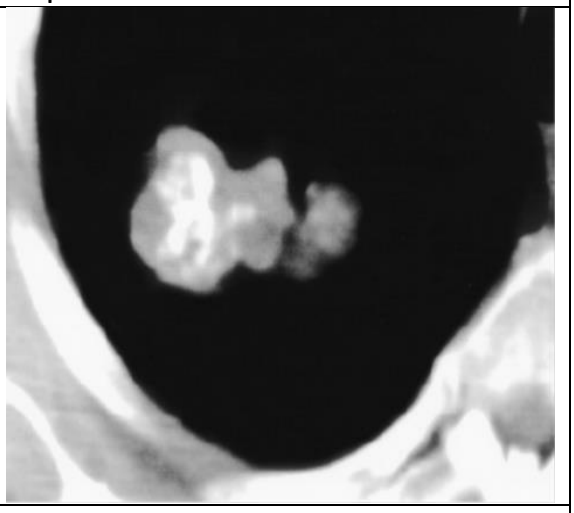
- Diffuse, central (bull's-eye appearance), laminated, and popcorn.
  - ⇨ Hamartoma – Granuloma



Granuloma = Central Calcification

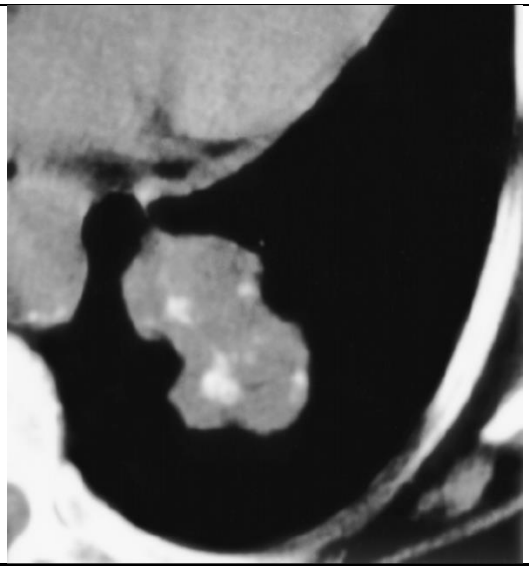
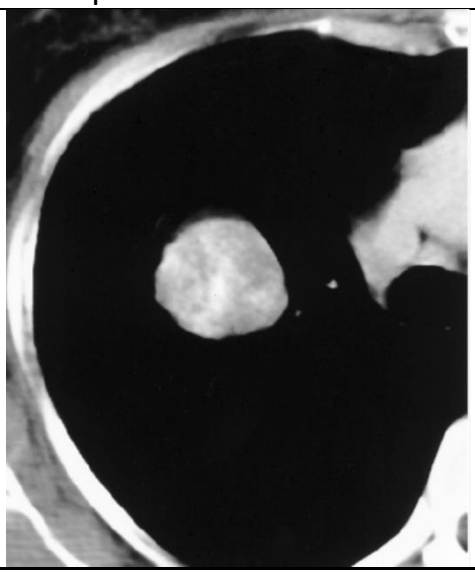


Popcorn Calcification



Indeterminate patterns of calcification:

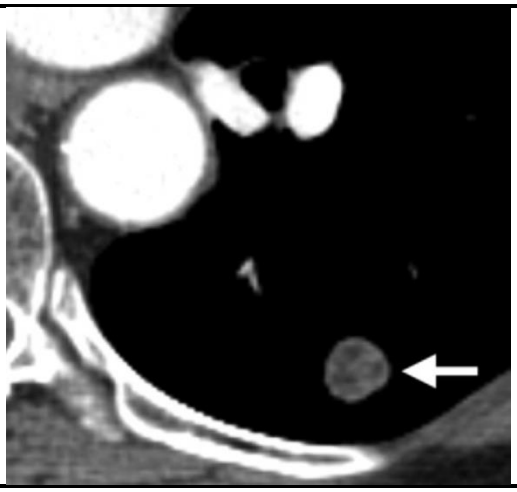
- Punctate, eccentric, and amorphous calcifications
- ⇨ Calcified metastasis: Osteosarcoma – chondrosarcoma – Mucinous cystadenocarcinoma of colon & ovary – Papillary cancer thyroid.
- ⇨ Carcinoid tumor
- ⇨ Adenocarcinoma

Carcinoid tumor Punctate calcification	Adenocarcinoma Amorphous calcification
	

5) Fat Attenuation

Nodule attenuation (-40 to -120 HU)

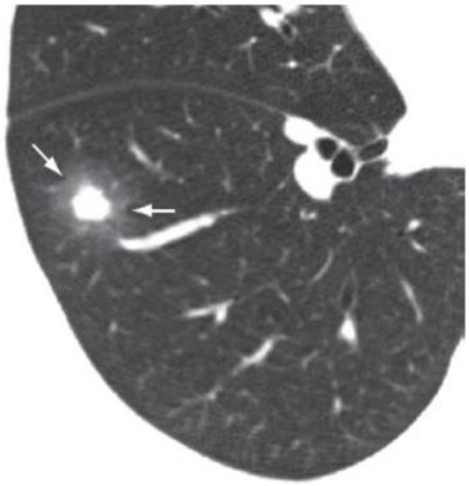
- ⇨ Hamartoma
- ⇨ Lipoid pneumonia
- ⇨ Pulmonary metastases from liposarcoma or renal cell cancer



6) Halo Sign

Poorly defined rim of ground-glass attenuation around the nodule.

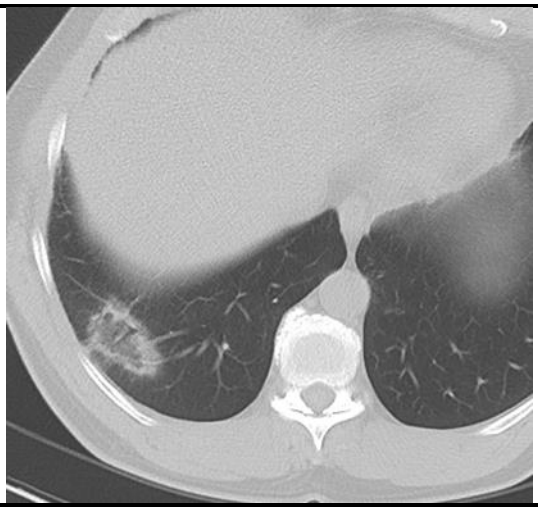
- ⇨ Hemorrhage
- ⇨ Tumor infiltration
- ⇨ Perinodular inflammation
- ⇨ Invasive aspergillosis
- ⇨ Adenocarcinoma in situ (Bronchioloalveolar carcinoma = BAC)
- ⇨ Kaposi sarcoma
- ⇨ Lung metastases from angiosarcoma, choriocarcinoma, osteosarcoma



7) Reverse Halo Sign = Atoll Sign

A central area of ground-glass attenuation surrounded by a halo or crescent of consolidation

- ⇨ Cryptogenic organizing pneumonia (COP)
- ⇨ Lung cancer after radiofrequency ablation



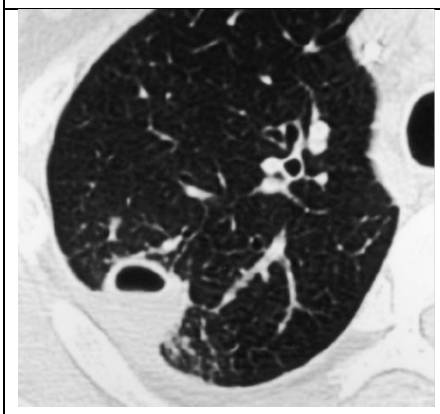
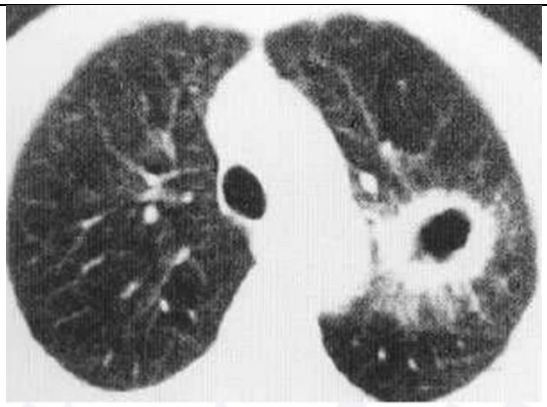
8) Cavitation

<u>Benign cavitary nodules:</u>

- Thin (<5mm), Smooth walls
- ⇨ Infectious and inflammatory conditions: Abscesses, infectious granulomas, vasculitides, and pulmonary infarction.

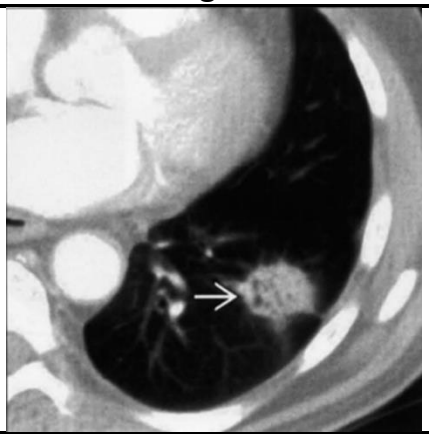
<u>Malignant cavitary nodule:</u>

- Thick (>15mm), irregular walls
- ⇨ Primary tumors
- ⇨ Metastatic, particularly squamous cell histologic characteristics.

Benign cavity Thin Smooth walls	Malignant cavity Thick-walled cavity Speculated outer surface Nodular inner surface
	

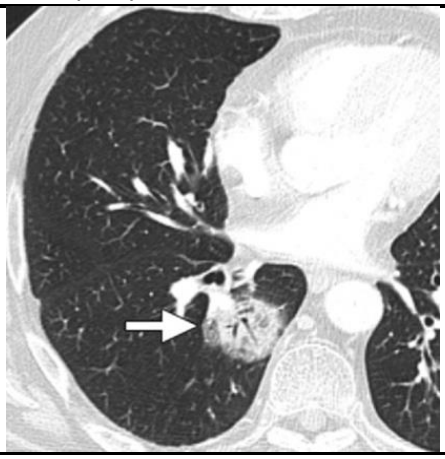
9) Pseudocavitation = Bubbly Lucencies

- Small focal low attenuation within or surrounding nodule.

⇨ Adenocarcinoma ⇨ Lymphoma ⇨ Sarcoidosis ⇨ Organizing pneumonia	

10) Air Bronchogram Sign

Malignant nodules (29%) > Benign nodules (6%)

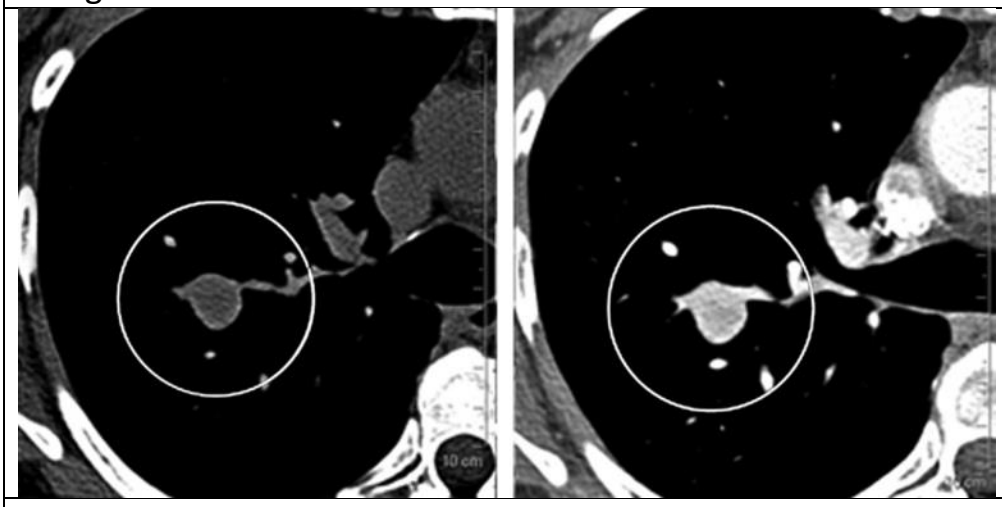
⇨ Adenocarcinoma ⇨ Lymphoma ⇨ Infection	

11) Nodule Growth

- Nodule volume = 4πr3.
- Doubling in volume = 26% increase in diameter.
- Much overlap between benign and malignant nodules
- Benign solid nodules => Volume doubling time <20 days or >400days
- Malignant solid nodules => Volume doubling time 20 –400 days
- 2-Year rule: No change in 2 years implies benignity

12) Nodule Enhancement:

Malignant nodules enhance >20 HU
Benign nodules enhance <15 HU

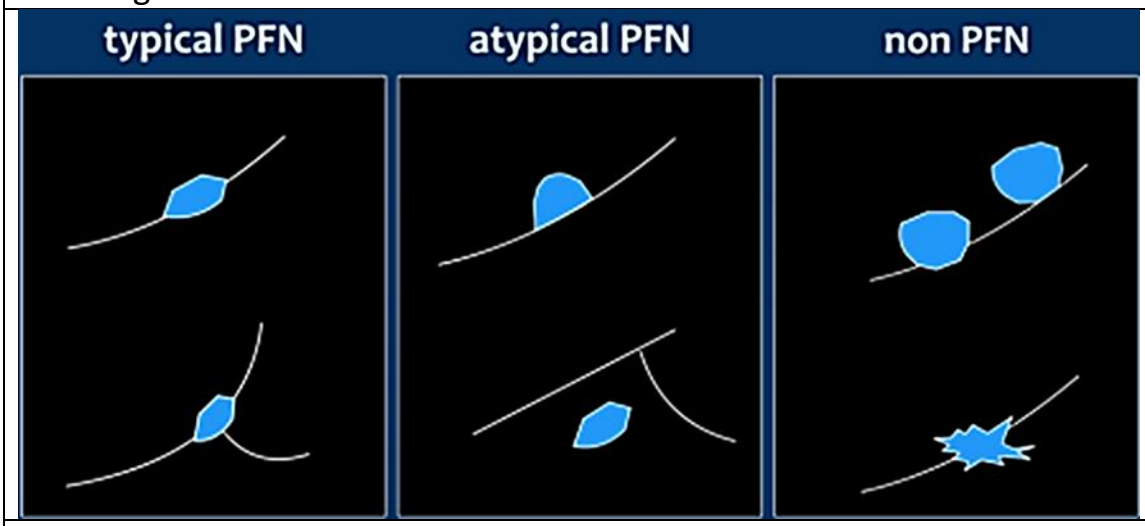


13) Nodule Metabolism:

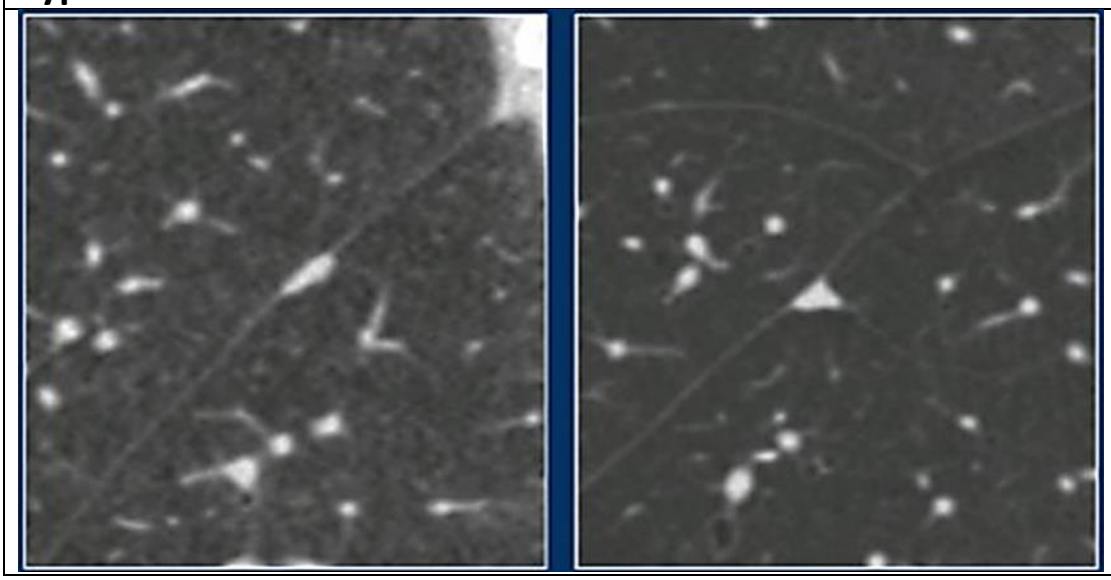
- SUV cutoff of 2.5 has been used to differentiate between benign and malignant nodules
- PET has sensitivity and specificity of approximately 90% for detecting malignant nodules with a diameter > 10 mm

❖ Perifissural Nodule (PFN)

- Attached to pulmonary fissure
- Homogenous
- Smooth margins
- Shaped: oval – lentiform – triangular
- Probaply intrapulmonary LN
- Needs no follow up.
- Benign



Typical PFN



❖ FLEISCHNER SOCIETY RECOMMENDATIONS 2013

❖ Fleischner Guidelines for Solid Nodules

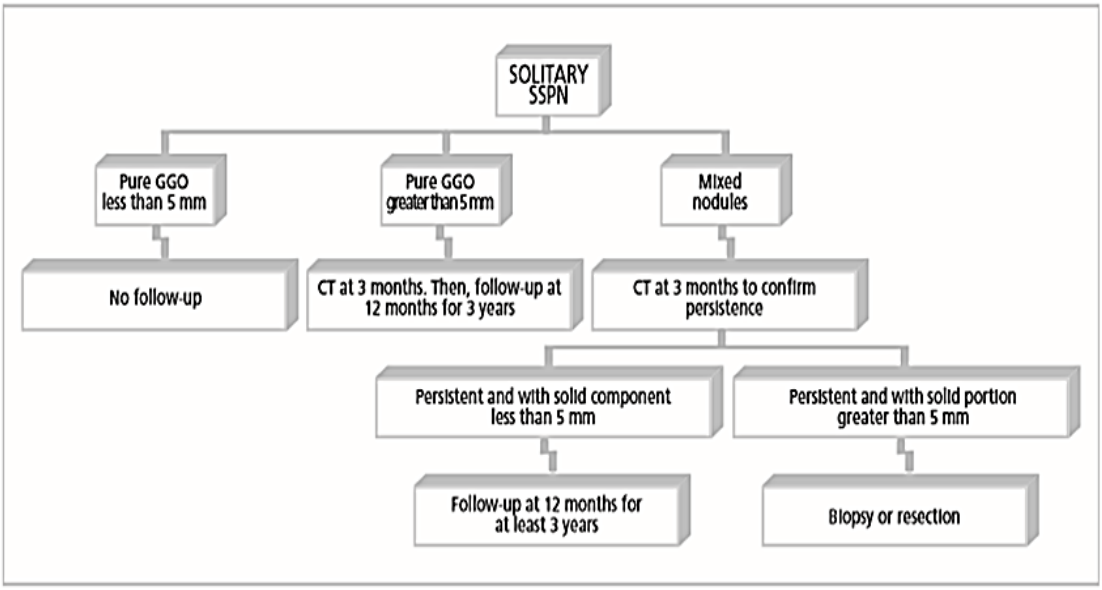
Solid	Average Size	Follow up	
	≤ 4 mm	Low risk	No Follow-up
		High risk	Follow-up 12 months Stop if unchanged
	4-6 mm	Low risk	Follow-up 12 months Stop if unchanged
		High risk	Initial FU 6-12 months 18-24 months if unchanged
	6-8 mm	Low risk	Initial FU 6-12 months 18-24 months if unchanged
		High risk	FU 3 / 9 / 24 months if unchanged
	> 8 mm	All	FU 3 / 9 / 24 months if unchanged or consider PET / Biopsy

Notes using Fleischner guidelines for Solid Pulmonary nodules

- Only apply in subjects 35 years or older.
- Subjects should be separated in low- and high-risk based on smoking characteristics.
- Do not apply in subjects with a known or suspected malignancy
- Measure the average of diameters in the axial plane
- Use thin-slice unenhanced (low-dose) scans to characterize and follow-up pulmonary nodules

❖ Fleischner Guidelines for Subsolid Nodules

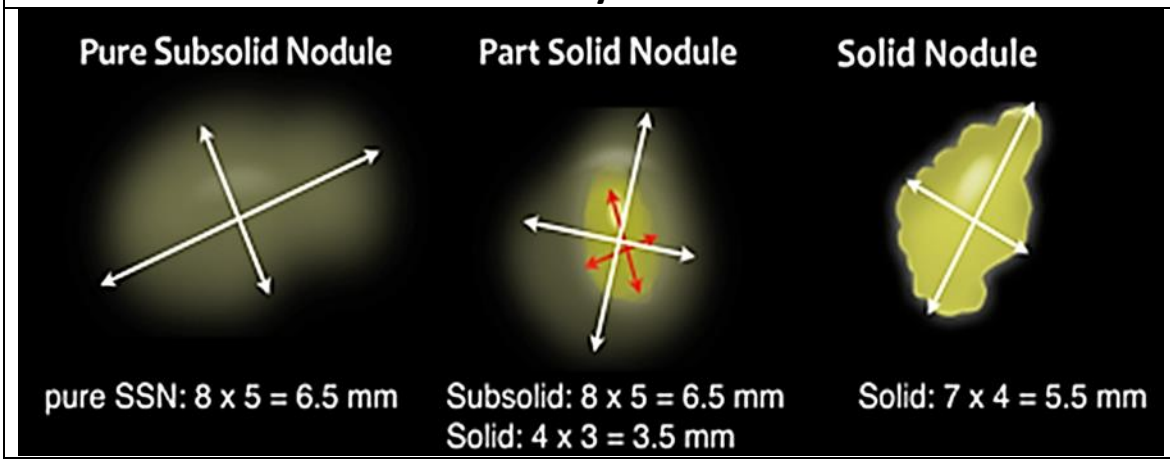
Subsolid	Average Size	Follow up
Subsolid	≤5mm	No FU indicated
	>5mm	Initial FU 3 months Persistent: annual for 3 years
Part-solid	≤5mm	Initial FU 3 months Persistent: annual for 3 years
	>5mm	Initial FU 3 months Persistent and solid component >5mm: ‣ Resection or biopsy
	>10mm	Consider PET
Multiple	≤5mm	FU 24 and 48 months
	>5mm	No dominant lesion: ‣ Initial FU 3 months ‣ Persistent: annual for 3 years
		Dominant lesion with part-solid component: ‣ Initial FU 3 months ‣ Persistent: resection or biopsy


SOLITARY SSPN
Pure GGO less than 5 mm
Pure GGO greater than 5 mm
Mixed nodules
No follow-up
CT at 3 months. Then, follow-up at 12 months for 3 years
CT at 3 months to confirm persistence
Persistent and with solid component less than 5 mm
Persistent and with solid portion greater than 5 mm
Follow-up at 12 months for at least 3 years
Biopsy or resection

Notes using Fleischner guidelines for Subsolid pulmonary nodules

- Apply in subjects of any adult age.
- No separation in low- and high-risk based on smoking.
- May be applied in subjects with a known or suspected malignancy
- Measure the average of diameters in the axial plane
- Use thin-slice unenhanced (low-dose) scans to characterize and follow-up pulmonary nodules

❖ **Measurement of Pulmonary Nodule**

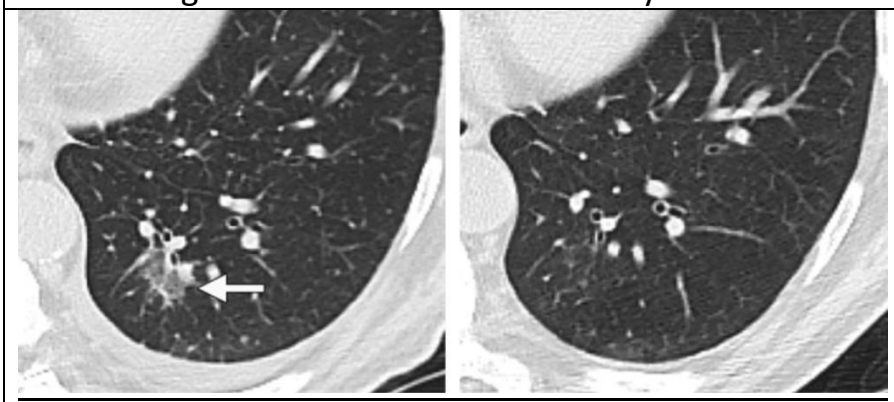


Subsolid Nodules (SSN)

Ground glass attenuation nodules (GGAN) or partly solid nodules
⇨ 3m follow up:

a) 3m follow up => Nodule regress or resolve:

⇨ Benign = infectious or inflammatory causes



b) Persistent nodules => long follow up for 3y:

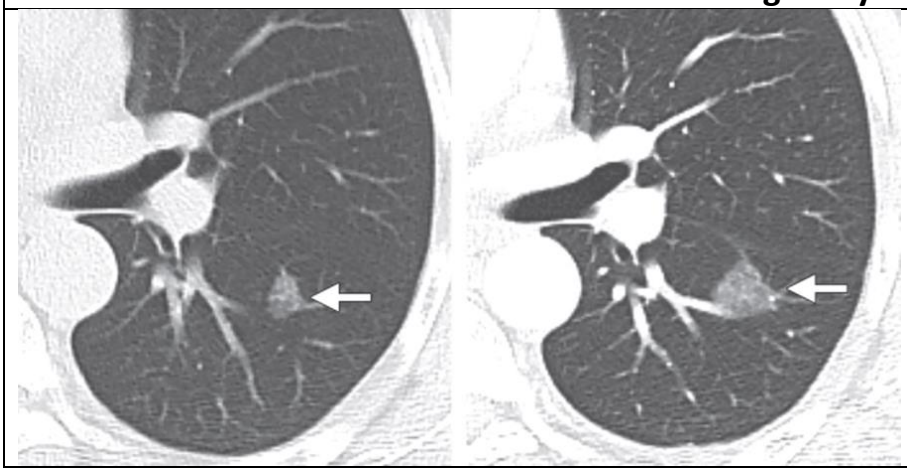
Signs of increased risk for malignancy:

⇨ Increase in size
⇨ Increase in attenuation
⇨ Increase in size of a solid component
⇨ Development of a solid component

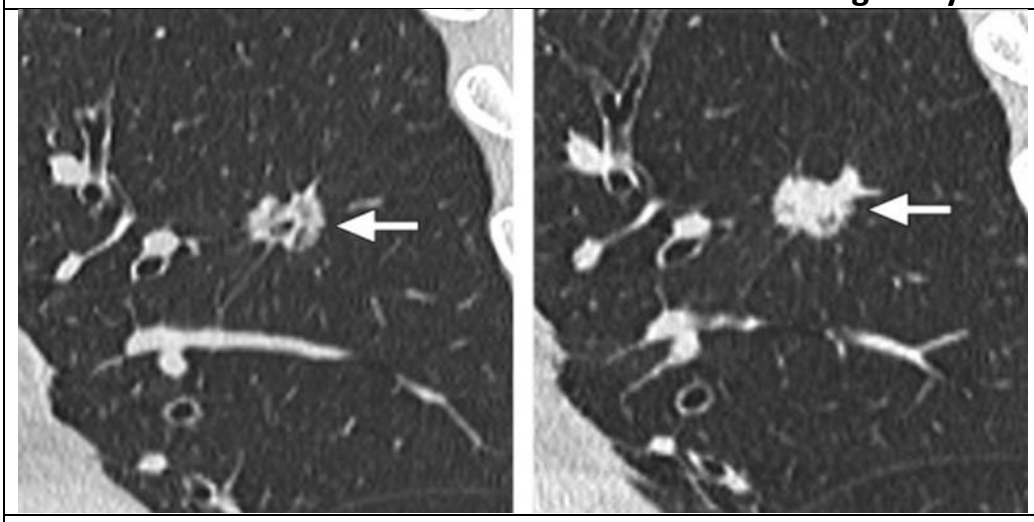
Increased risk for malignancy (Adenocarcinoma):

⇨ Atypical adenomatous hyperplasia (AAH)
⇨ Adenocarcinoma in-situ (AIS)
⇨ Minimally invasive adenocarcinoma (MIA)
⇨ Invasive adenocarcinoma

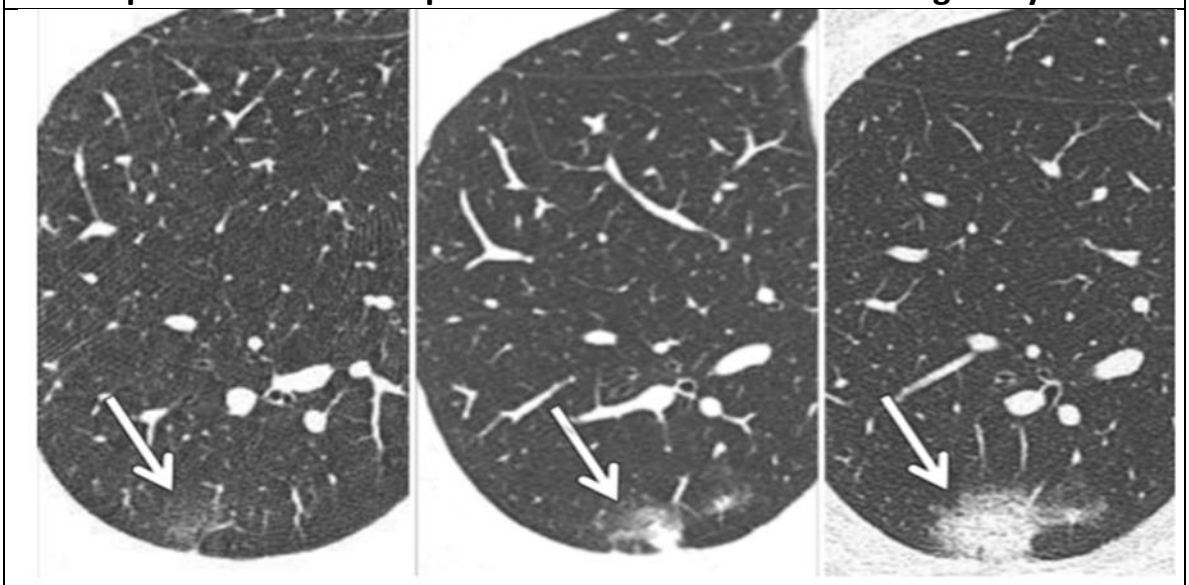
Subsolid nodule (GGAN) => 3m follow up =>
nodule increase in size => increased risk for malignancy



Subsolid nodule (GGAN) => 3m follow up =>
nodule increase attenuation => increased risk for malignancy.



Subsolid nodule (GGN) => 3m follow up =>
Development of solid component => increased risk for malignancy.

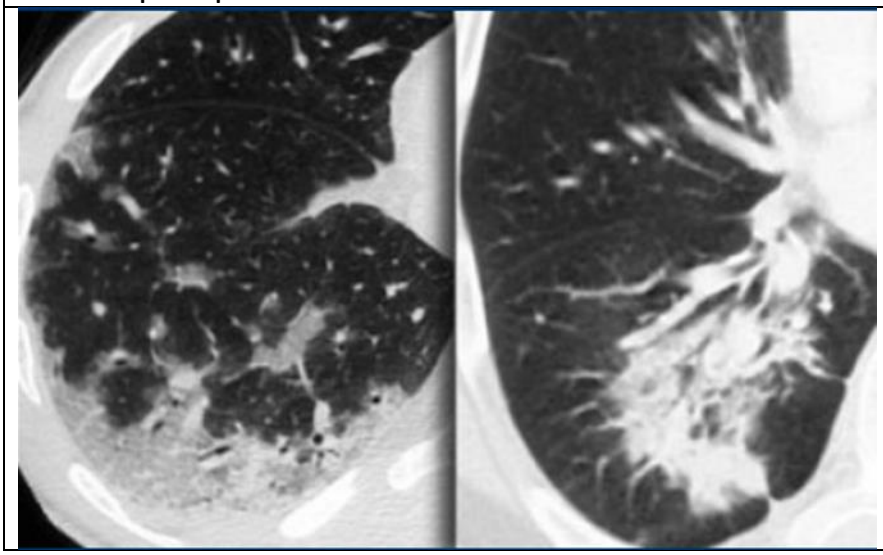


B- Pulmonary Consolidation

- Air in acini is replaced by fluid or tissue.
- Air bronchogram = Represents a lucent air-filled bronchus (or bronchiole) seen within consolidation.
- No volume loss.
- Pulmonary vessels are not visible through the consolidation on an unenhanced CT.
- Non-segmental distribution = Intersegmental spread: due to channels of collateral drift allow passage of air and fluid:
    - Pores of Kohn (Inter-alveolar openings)
    - Channels of Lambert (Bronchoalveolar communications)
    - Direct airway anastomosis (120um in diameter)

Causes of Consolidation:

1. Lobar pneumonia
2. Pulmonary contusion / hemorrhage
3. Pulmonary Infarction
4. Bronchoalveolar carcinoma
5. Lymphoma
6. Radiation pneumonitis
7. Lipoid pneumonia



C- Solitary Pulmonary Cavity

Pattern:

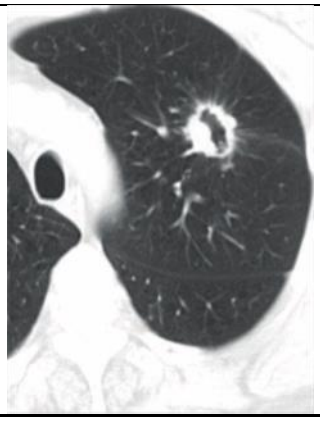
- Air-filled space surrounded by complete wall
- Wall thickness > 3mm

Causes:

Infection: Lung abscess – TB – Fungal ball – Hydatid cyst

Neoplastic: Bronchogenic carcinoma

	TB Cavity	Malignant Cavity
Wall thickness	≤ 4mm	> 15mm
Inner surface	Smooth	Irregular / Nodules / Solid mural component
Surrounding	Fibrosis	Speculated margins

Malignant Cavity	Post-Primary TB Cavity	Fungal Ball
		

D- Solitary Pulmonary Cyst

Pattern:

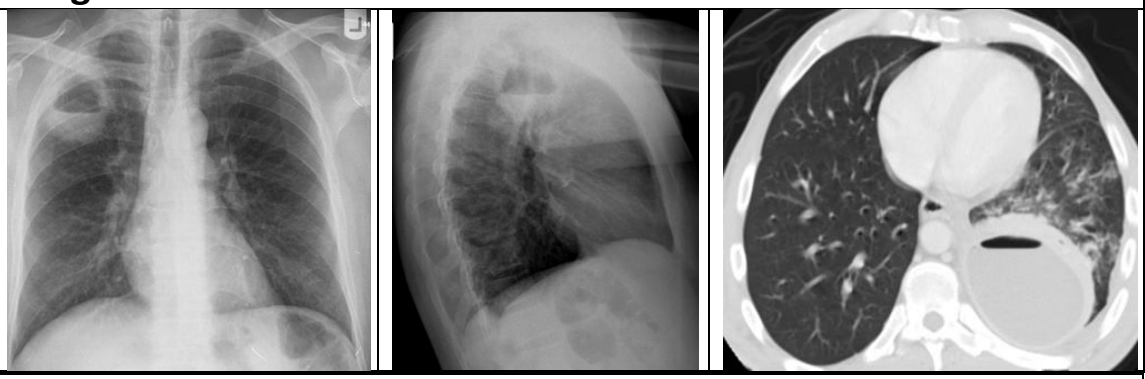
- Air-filled space surrounded by complete wall
- Wall thickness < 3mm

Causes:

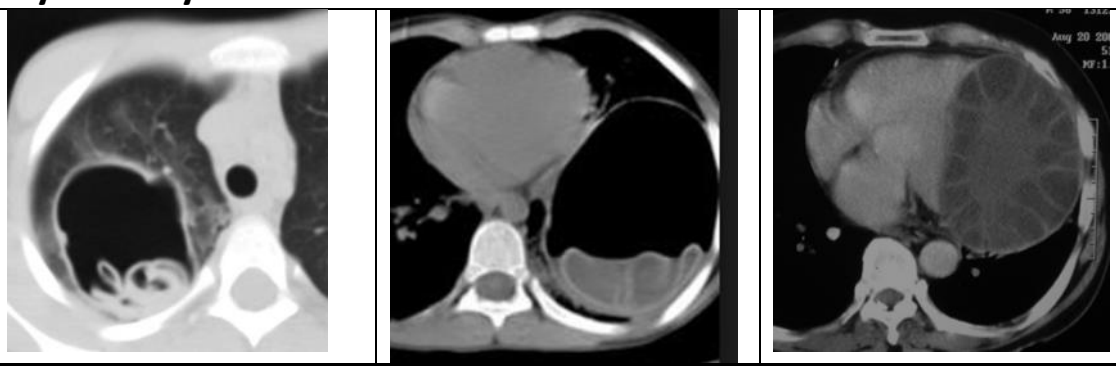
1) Emphysematous Bulla (>1cm) / Bleb (<1cm)
2) Pneumatocele (caused by trauma)
3) Infection: Lung abscess - Hydatid cyst
4) Congenital: Bronchogenic cyst – CCAM

	Abscess	Hydatid Cyst
Wall thickness	Thick enhancing	Thin non-enhancing
Content	Fluid density Air / fluid level Air density	Fluid density Water lily sign Meniscus sign
Surrounding	Consolidation	Normal lung

Lung Abscess



Hydatid Cyst



E- Atelectasis = Collapse

Atelectasis is loss of lung volume due to decreased aeration.

CXR & CT Findings:

A. Direct signs:

1) Increased opacity / density
2) Volume loss
3) No air-bronchogram
4) Vascular crowding
5) Displacement of the fissures.

B. Indirect signs:

6) Elevation of the diaphragm.
7) Rib crowding on the side with volume loss.
8) Mediastinal shift to the side with volume loss.
9) Hilar displacement to the side with volume loss.
10) Overinflation of adjacent or contralateral lobes.

Lobar atelectasis is usually caused by central bronchial obstruction (obstructive atelectasis), which may be secondary to mucus plugging or an obstructing neoplasm.

Subsegmental atelectasis is caused by obstruction of small peripheral bronchi, usually by secretions. Air bronchograms can be seen

Types of atelectasis:

a) Obstructive (Resorpitive) atelectasis: caused by bronchial obstruction:
   i. Intrinsic (Endobronchial) lesion: FB – Mucus plug – Metastasis
   ii. Extrinsic: Tumor – LNs (Broncholithiasis)
b) Relaxation (passive) atelectasis: caused by pleural effusion, pneumothorax or pulmonary mass.
c) Adhesive atelectasis: due to surfactant deficiency (ARDS).
d) Cicatricial atelectasis: due to architectural distortion of lung parenchyma by fibrosis (TB-Sarcoidosis-Silicosis)
e) Round atelectasis: seen is pleural disease (Asbestosis)

LUL Collapse

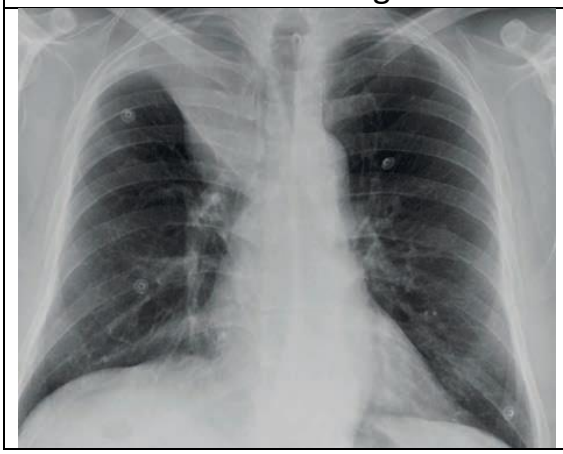
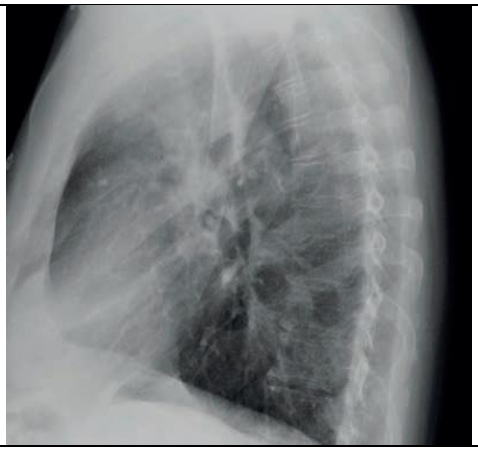
PA	Lateral
Left upper hazy density	Apical wedge shaped density Anterior displacement of the left oblique (major) fissure

Luftsichel sign: A crescent of air lateral to the aortic arch representing upward migration of the apical segment of the left lower lobe.

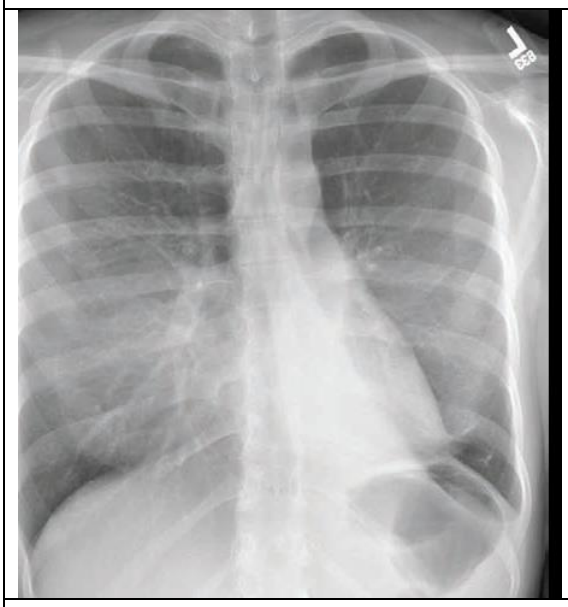
RUL Collapse

Right upper opacity Elevation of minor fissure Tracheal shift to the right side Elevation of the right hilum	Wedge shaped apical opacity Anterioir displacement of the major fissure

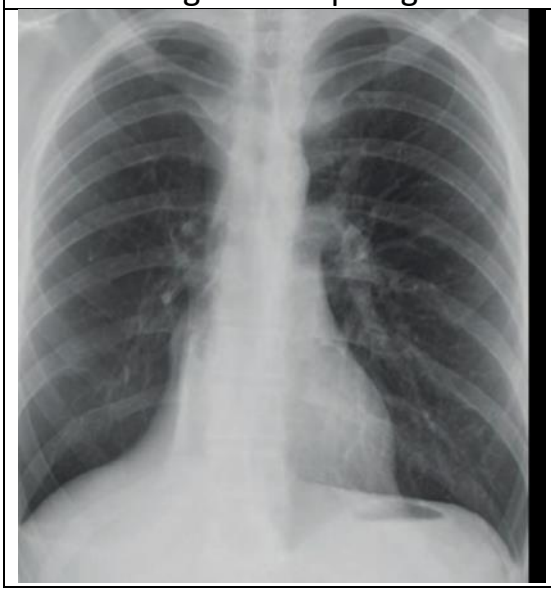
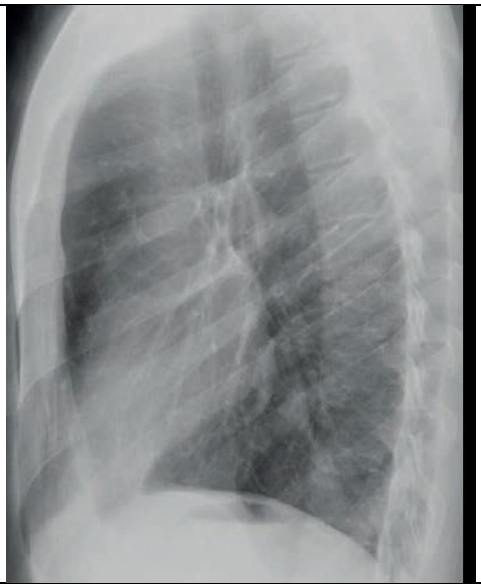
Golden's S sign: Right upper lobe collapse caused by an obstructing mass. The central convex margins of the mass form a reverse S.

LLL Collapse

Left retro-cardiac triangular density Silhouetting with diaphragm	Wedge-shaped opacity projecting over the heart
Flat waist sign: flattening of the left heart border as a result of downward shift of hilar structures and resultant cardiac rotation	
	

RLL Collapse

Right retro-cardiac triangular density Silhouetting with diaphragm	Wedge-shaped opacity projecting over the heart
	

RML Collapse

indistinct opacity in right lung Silhouetting of right heart border	Wedge-shaped opacity (arrow) projecting over the mid-heart

RML Syndrome:

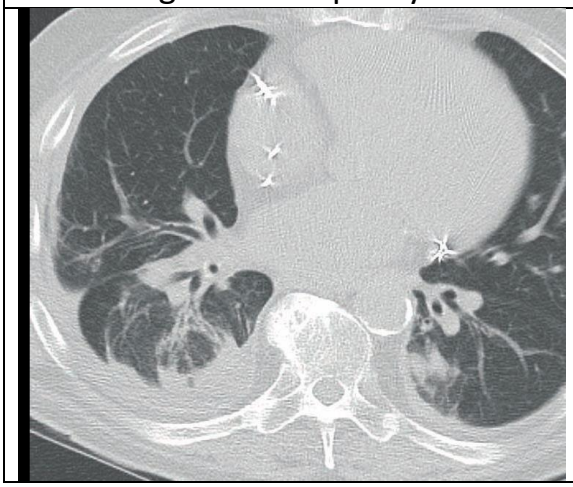
Bronchial obstruction by calcified hilar LNs (Broncholithiasis) or granuloma (TB – Silicosis – Histoplasmosis)

Recurrent atelectasis – consolidation – bronchiectasis.

Round Atelectasis

most common in the posterior lower lobes

1) Abnormal pleural: pleural thickening – effusion – plaques
2) Peripheral pleural based opacity
3) Rounded / elliptical opacity
4) Volume loss in the affected lobe.
5) Comet tail sign = curved pulmonary vessels and bronchi from hilum leading into the opacity



II- Diffuse Lung Patterns

Patterns:
1) Multiple Nodules 2) Multiple Cysts 3) Multiple Cavitary Nodules 4) Reticular – Reticulonodular 5) Diffuse Consolidation / Ground Glass Opacities (GGO)
Approach:
1) Pattern: Reticular – Reticulonodular – GGO – Nodules – Cysts 2) Distribution: Upper vs Lower & Central vs Peripheral 3) Lung volume: Normal / Increased / Decreased 4) Evolution: Acute / Chronic 5) Pleural disease. 6) Mediastinal / hilar LNs 7) Associations

A- Nodular Pattern

A) Centrilobular Nodules

- Opacification of the centrilobular bronchiole (or less commonly the centrilobular artery)
- At the center of each 2ry pulmonary lobule + sparring lung periphery
- Solid or ground glass nodules

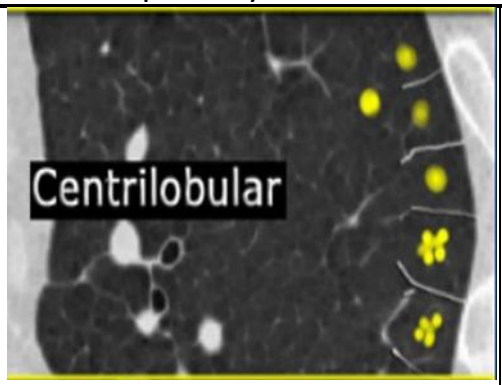
Causes:

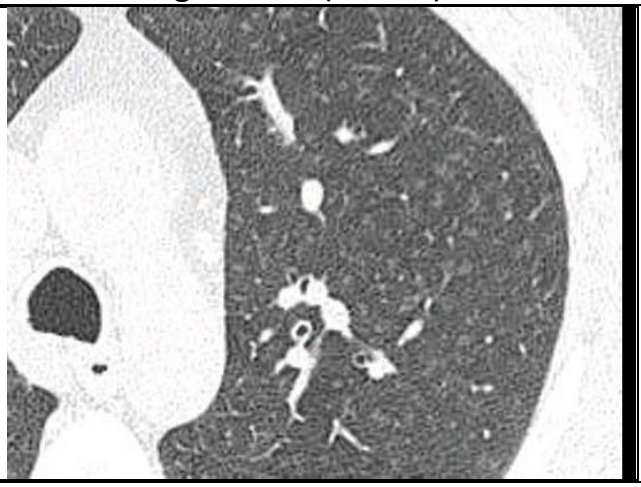
I) Infectious causes:

Endobronchial spread of: TB - MAI
Bronchopneumonia

II) Inflammatory causes:

Hypersensitivity pneumonitis (HSP)
Respiratory bronchiolitis interstitial lung disease (RB-ILD)





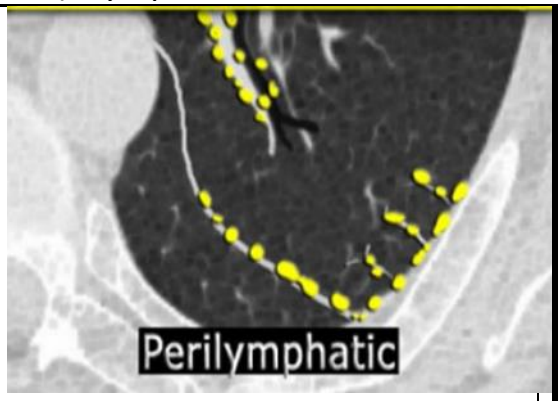
B) Perilymphatic Nodules

follow the anatomic locations of pulmonary lymphatics:

1. Subpleural.
2. Peribronchovascular.
3. Interlobular Septal

Causes: (2S + 3L)

1) Sarcoidosis
2) Silicosis - Coal workers pneumoconiosis
3) Lymphangitis carcinomatosis
4) Lymphocitic interstitial pneumonia
5) Lymphoma

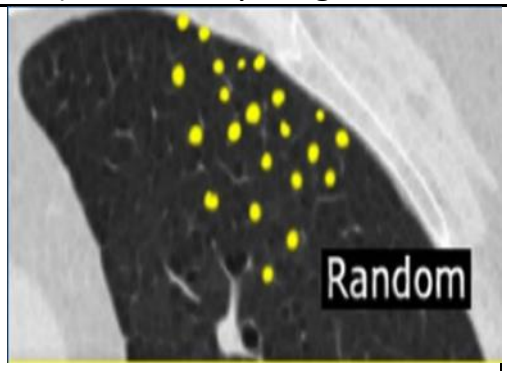


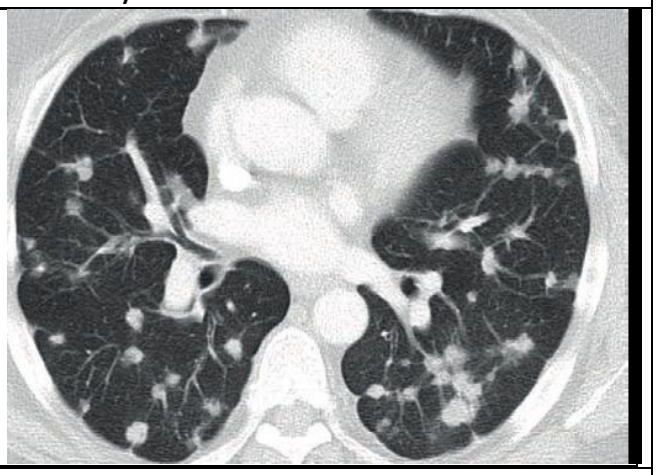
C) Random Nodules

occur via hematogenous spread and have an angiocentric distribution

Causes:

1) Hematogenous metastases.
2) Septic emboli.
3) Pulmonary Langerhans's cell histiocytosis



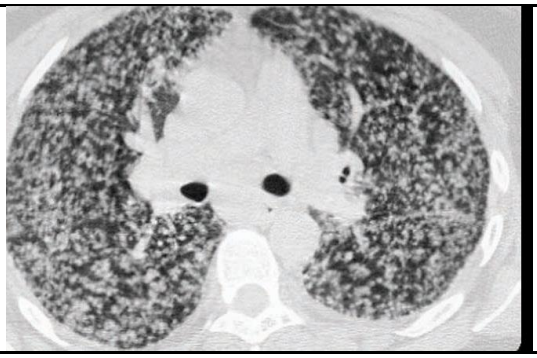


D) Miliary Nodules

Innumerable bilateral tiny random pulmonary nodules

Causes:

1) Miliary TB - Histoplasmosis.
2) Miliary fungal infection.
3) Miliary metastases: thyroid – Melanoma – Choricarcinoma.



E) Calcified Nodules

Causes:

1) Metastases: Medullary thyroid carcinoma – Mucinous – Osteogenic
2) Infection: Varicella – TB – Histoplasmosis – Schistosomiasis
3) Silicosis – CWP
4) Alveolar Microlithiasis
5) Chronic pulmonary venous hypertension
6) Severe renal disease

F) Tree in-Bud:

- Multiple small nodules + connected to linear branching structures
- Centrilobular location
- Represent dilated and fluid filled centrilobular bronchioles

Causes:

I) Infectious causes:

Bacterial: Endobronchial spread of:
- Mycobacterium tuberculosis
- M. avium-intracellulare complex
- Staph. aureus bronchopneumonia

Fungal
- Aspergillus (Airway-invasive aspergillus)

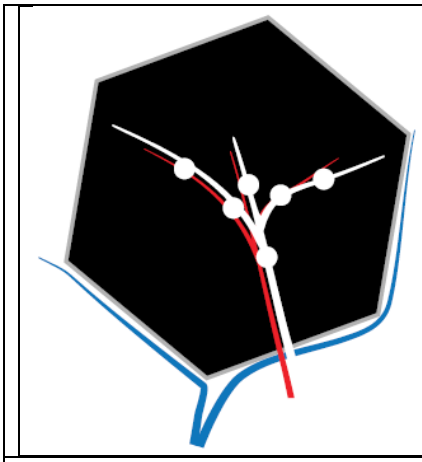
Viral
- Cytomegalovirus
- Respiratory syncytial virus

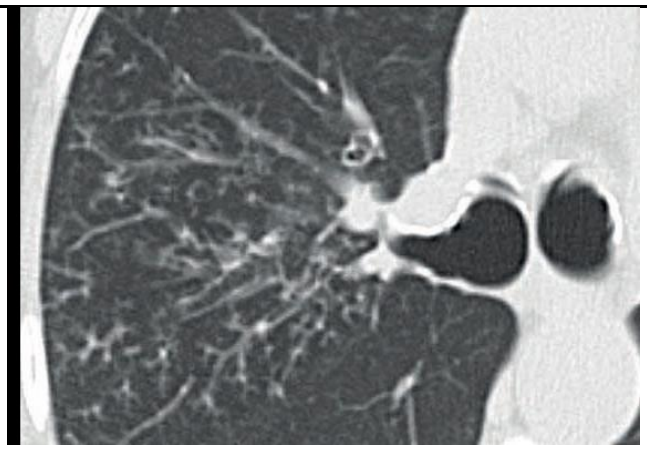
II) Inflammatory causes:

Hypersensitivity pneumonitis (HSP)
Respiratory bronchiolitis interstitial lung disease (RB-ILD)
Obliterative bronchiolitis = Bronchiolitis obliterans
Aspiration
Inhalation of toxins
Connective tissue disorders
- Rheumatoid arthritis
- Sjogren syndrome

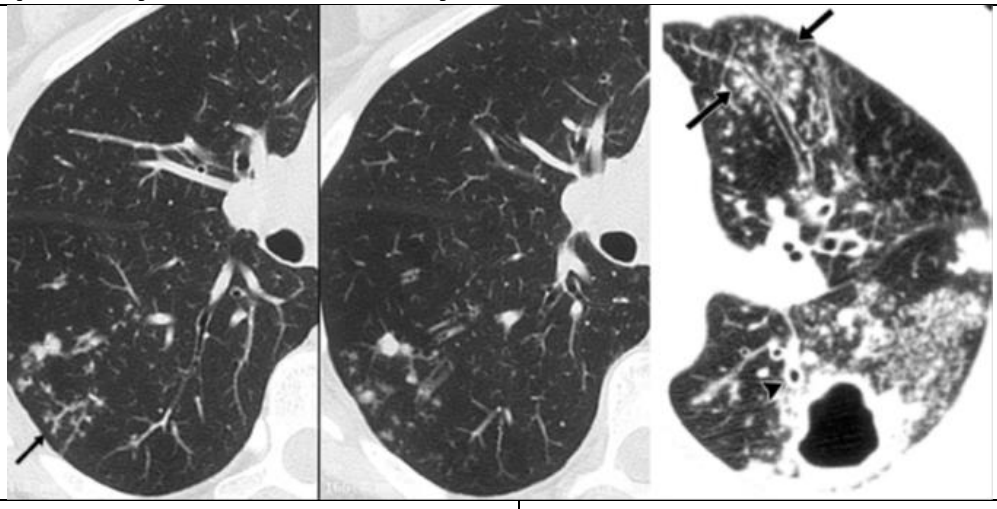
III) Congenital:

Cystic fibrosis
Primary Ciliary Dyskinesia = Immotile Cilia Syndrome
- Kartagener Syndrome

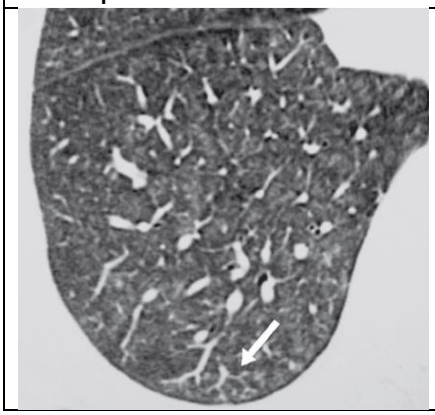




Post-primary active TB: Cavity + tree-in-bud nodules



CMV pneumonia

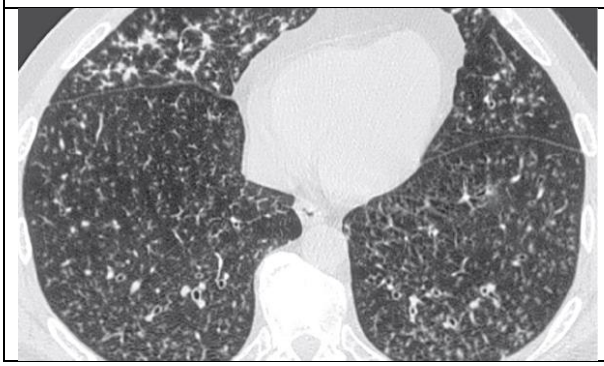


RSV pneumonia

Kartagener Syndrome:

Situs inversus + Chronic sinusitis + Bronchiectasis + Tree-in-bud

Obliterative Bronchiolitis



B- Reticular Pattern

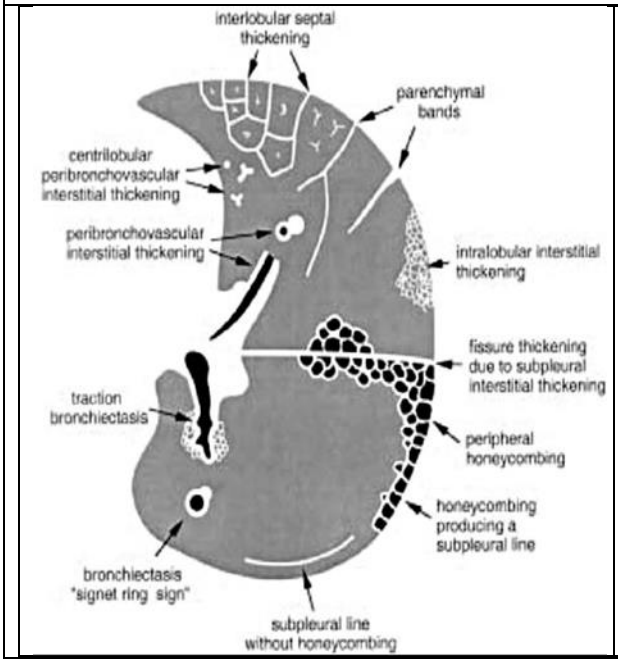
A) Smooth Interlobular Septal Thickening

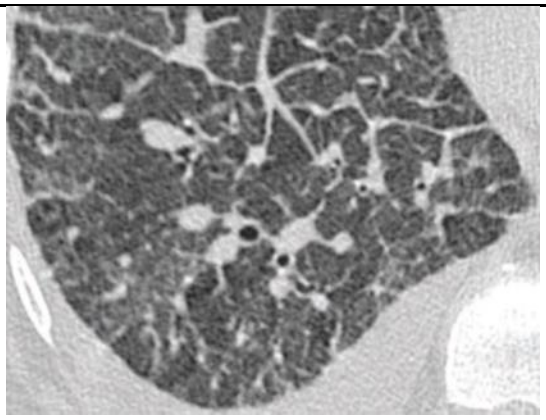
Causes:

1) Pulmonary edema
2) UIP (IPF) – NSIP
3) Asbestosis
4) Collagen Vascular disease: RA – Scleroderma
4) Drugs – Radiation
5) Extrinsic Allergic Alveolitis = Hypersenstivity Pneumonitis (Chronic phase)

Pattern:

- Peribronchovascular thickening / cuffing (Karley's A lines = Central)
- Interlobular septal thickening (Karley's B lines = Peripheral)
- Intralobular interstitial thickening
- Fissural thickening.
- Subpleural curvilinear lines
- Parenchymal bands
- Traction bronchiectasis
- Honeycombing = Subpleural cysts
- Late => Architectural distortion.





	Interstitial Pulmonary Edema	UIP = IPF	NSIP	Asbestosis	Scleroderma
Pattern	Karley's A lines Karley's B lines	Septal thickening Honeycombing Traction bronchiectasis	GGO Septal thickening	Septal thickening Honey combing Traction bronchiectasis	GGO Septal thickening
Distribution	Central & Peripheral	**Bi Basal & Peripheral Reticular Pattern**			
Lung Volume		**Late Decreased**			
Pleural disease	Pleural effusion	Pl thickening		Pl thickening Pl effusion Pl plaques Pl calcification Round atelectasis	
LNs	-ve				
Associations	Cardiomegaly			Malignancy: Mesothelioma Bronchogenic Ca GI Neoplasms	CREST: Calcinosis Raynaud's Esophageal dilatation Sclerodacty Telangiectasia

	HP / EAA	TB	Sarcoidosis	Silicosis / CWP
Pattern	Acute phase: GGO/Consolid Centrilobular nodules Mosaic attenuation + Air trapping (headcheese sign)	Tree-in-bud nodules Exudative pattern: Patchy or confluent consolidation Fibro-cystic pattern: TB Cavity ± Fungal ball (Aspergilloma)	Reticulo-nodular pulmonary infiltration with lymphatic distribution	Nodular pattern Calcified nodules 20% Reticulo-nodular pattern Progressive massive fibrosis
	Chronic phase: Pulmonary fibrosis **Honeycombing – Traction bronchiectasis** **Hilar retraction + Mediastinal shift** **Paracicatricial emphysema – Bullae – Fungal ball**			
Distribution	**Bi UL Pulmonary Fibrosis**			
Lung Volume	**Late Lung Volume Decreased**			
Pleural disease	**Apical pleural thickening**			
LNs		No significant LNs	Bi hilar & mediastinal LNs	Egg shell calcification

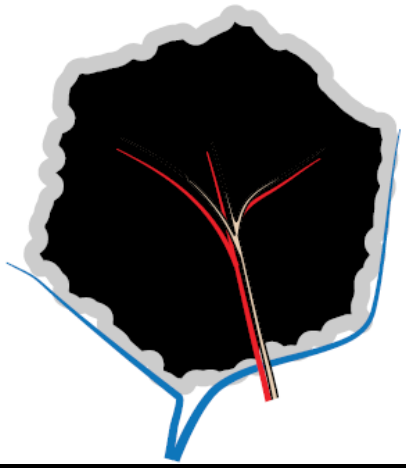
B) Nodular Septal Thickening:

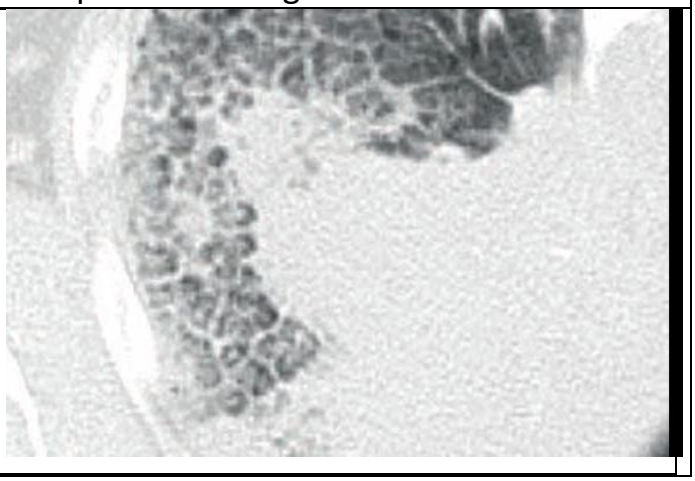
Causes:

1) Sarcoidosis
2) Silicosis – CWP
3) Lymphangitis Carcinomatosis
4) Lymphocytic Interstitial Pneumonia
5) Lymphoma

Pattern => Perilymphatic distribution:

Peribronchovascular nodular thickening
Irregular and nodular interlobular septal thickening





	Sarcoidosis	Silicosis – CWP	Lymphangitis Carcinomatosis	Lymphoma
Pattern	**RN pattern**			
Distribution	Upper & Middle lung zones	Upper lung zones		
Lung Volume	Normal or Increased			
LNs	Bi&Sym Hilar LN Paratracheal LN	Hilar LN Egg shell Ca	Malignant LNs	Hilar & Mediastinal LNs
Pleural disease	Pl effusion (10%)		Malignant Pl effusion	Pleural effusion doesn't contain maliganant cells
Associations	Bilateral upper fibrosis – Pl thickening – Traction bronchiectasis – Bullae – TB cavity – Fungal ball Caplan Syndrome = RA + Silicosis/CWP			• Bronchovascular form. • Pneumonic form • Subpleural form • Nodular form

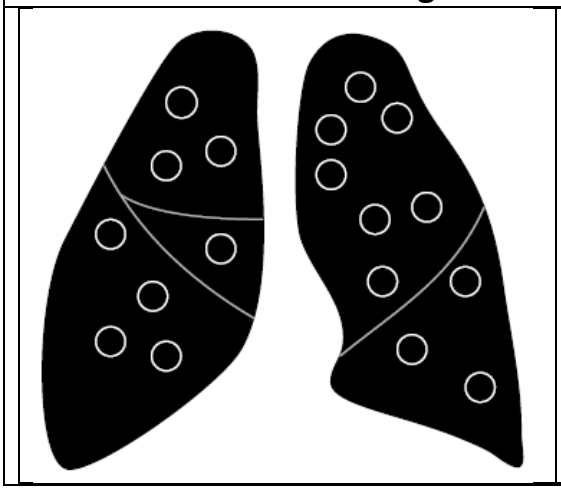
C- Multiple Cysts

Causes: 3L

1) **Langerhans Cell Histocytosis (LCH)**
2) **Lymphangioleiomyomatosis (TS)**
3) **Lymphocytic Interstitial Pneumonia (LIP)**

4) **Cystic Bronchiectasis**
5) **Emphysema**

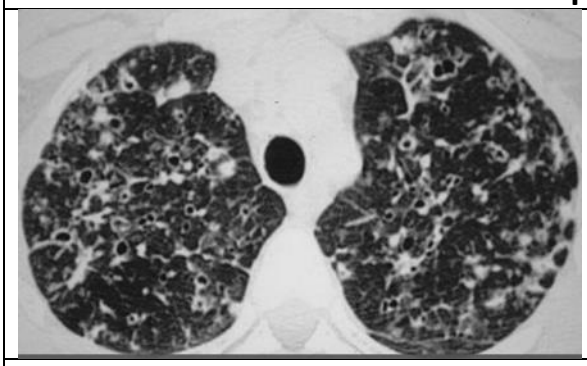
6) **Honey combing (Lung fibrosis):**
   - → **IPF = UIP**
   - → **Asbestosis**
   - → **Collagen vascular disease (RA – Scleroderma)**
   - → **Extrinsic allergic alveolitis**

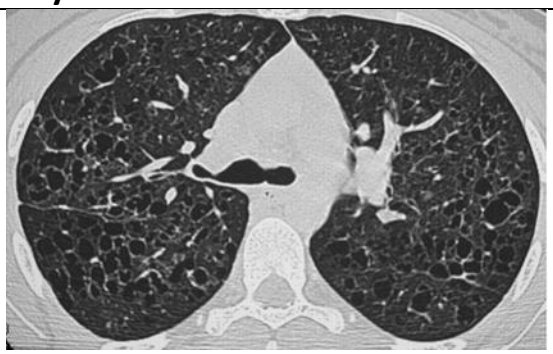


	Langerhans Cell Histocytosis (LCH)	**Lymphangioleiomyomatosis (LAM)**
	M > F Middle age Smoking related lung disease	Females Middle age Tuber sclerosis (TS) (Triad = Seizures +Mental Retardation + Adenoma sebaceum)
Pattern	Centrilobular nodules : Star fished shape Cysts: Bizarre shaped Normal intervening lung parenchyma	No nodules Cysts: Regular shapes & size Normal intervening lung parenchyma
Distribution	Upper zones Sparing lung bases	Whole lung
Lung Volumes	Normal or Increased	
Pleural disease	Spontaneous Pneumothorax	Recurrent pneumothorax Chylous pleural effusion

Pulmonary LCH

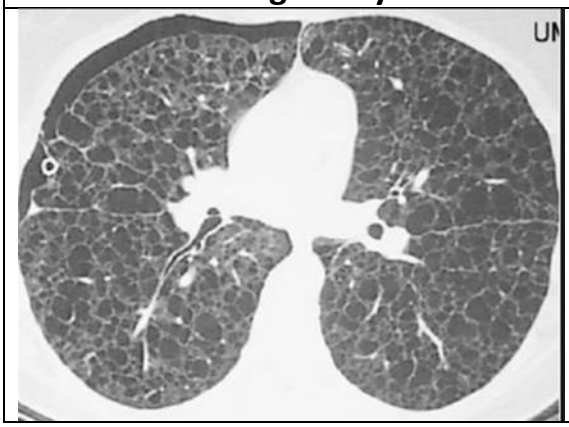
Nodules + variable sized bizarre shaped cysts





Pulmonary LAM

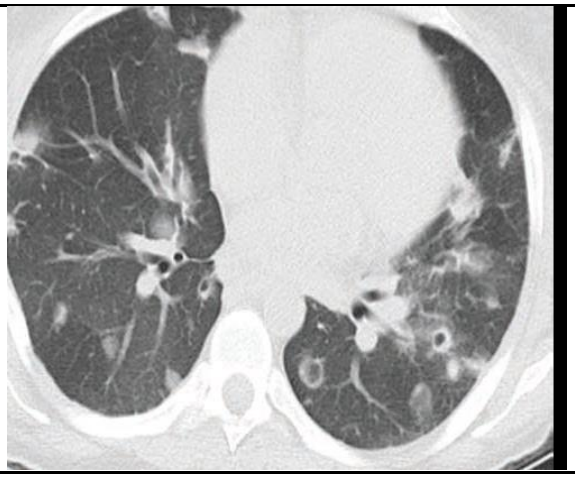
No nodules + Regular cysts + Pneumothorax + Chylous effusion



D- Multiple Cavitary Nodules

Causes:

1) TB – Fungal
2) Septic emboli.
3) Wegener granulomatosis
4) Metastases: Squamous cell carcinoma and uterine carcinosarcoma
5) Rheumatoid nodules



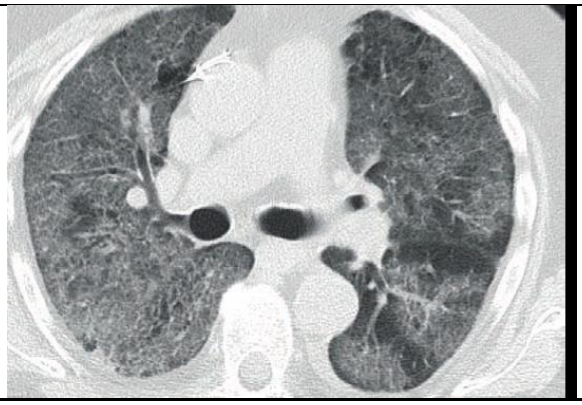
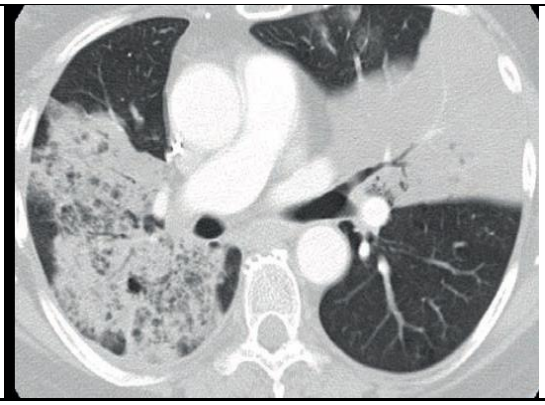
E- Diffuse Consolidation – Ground Glass Opacities

Consolidation:

- Air in acini is replaced by fluid or tissue.
- Air bronchogram = Represents a lucent air-filled bronchus (or bronchiole) seen within consolidation.
- No volume loss.
- Pulmonary vessels are not visible through the consolidation on an unenhanced CT.
- Non-segmental distribution = Intersegmental spread: due to channels of collateral drift allow passage of air and fluid:
    - Pores of Kohn (Inter-alveolar openings)
    - Channels of Lambert (Bronchoalveolar communications)
    - Direct airway anastomosis (120um in diameter)

Ground Glass Opacity = GGO

- Increased attenuation of lung
- Preservation of bronchovascular markings
- Geographic distribution
- Minimal septal thickening
- Physiologic ground-glass attenuation can be seen in the dependent lung areas: caused by a gravity-related increase in perfusion and decrease in amount of intra-alveolar air.

GGO	Consolidation
	

Acute vs Chronic? & Central vs Peripheral?

Acute + Central Consolidation / GGO = Batwing:

1) Pulmonary edema = Cardiogenic pulmonary edema
2) Diffuse pulmonary hemorrhage
3) Acute respiratory distress syndrome (ARDS)
4) Acute Interstitial pneumonia (AIP)
5) Acute Eosinophilic Pneumonia (AEP)

Chronic + Central consolidation / GGO:

1) Pulmonary Alveolar Proteinosis (PAP)
2) Hypersensitivity pneumonitis (HA=EAA) (Acute stage)
3) Pneumocystitis cranii pneumonia (PCP)
4) CMV in lung transplantation

Chronic + Peripheral Consolidation / GGO = Reverse Batwing:

1) Organizing pneumonia (COP = BOOP)
2) Eosinophilic lung disease:
    - Chronic eosinophilic pneumonia (CEP)
    - Loeffler's syndrome
    - Churg-Strauss Syndrome (CSS)
3) Bronchoalveolar carcinoma (BAC)
4) Pulmonary infarctions

Crazy Paving Pattern:

- Geographical distribution of ground glass opacities (GGO)
- Interlobular septal thickening
- No air-bronchogram

1) Pulmonary alveolar proteinosis (PAP)
2) Bronchoalveolar carcinoma (BAC)
3) Pneumocystis jiroveci pneumonia (PCP).
4) Organizing pneumonia (COP).
5) Lipoid pneumonia
6) Acute respiratory distress syndrome (ARDS).
7) Pulmonary hemorrhage.

Pulmonary Edema	ARDS	Pulmonary Hemorrhage	HP = EAA	PAP	PCP
LVF MS / MR Lt atrial thrombus / Myxoma	Toxin inhalation Drowning Mendelssohn's Syndrome Sepsis Burns	Pulm vasculitis SLE Idiopathic pulmonary hemosiderosis	Farmer's lung Bird fancier's ling	Idiopathic Drug induced Occupational	AIDS patients CD4 counts <200 cells/cc
Grade 1: Cephalization Grade 2: Interstitial edema Grade 3: Batwing alveolar edema	Consolidation GGO Gravity dependent	Consolidation GGO Crazy paving pattern	Acute phase: GGO – Consolidation Subacute phase: Centrilobular nodules	Crazy paving	GGO Centrilobular nodules Crazy paving
	No Karely's Lines No pulm fibrosis	Repeated attacks => Pulm fibrosis	Chronic phase => Pulm fibrosis	No Karely's lines No pulm fifbrosis	Upper lobe cysts /pneumatocele
Distribution : Bilateral + Central + Perihilar					
Cardiomegaly	**No Cardiomegaly**				
Pleural effusion	**No pleural effusion**				**Pneumothorax**

COP	CEP	CSS	BAC
Interstitial lung disease	Eosinophilic lung disease	Small vessel pulm vasculitis	Invasive mucinous adenocarcinoma (Mucinous BAC)
Idiopathic Collagen vascular diseases Drugs	Asthma + Peripheral eosinophilia (Ig E)	Asthma+ peripheral eosinophilia + P-ANCA is positive	Solid & subsolid nodule Multifocal/ Multilobar consolidation
Reverse halo = Atoll sign: Central lucency is surrounded by a peripheral opacity.		Fleeting opacities	Mass like consolidation
Pattern: Airspace opacities = Consolidation + GGO			
Distribution: Bilateral peripheral patchy			

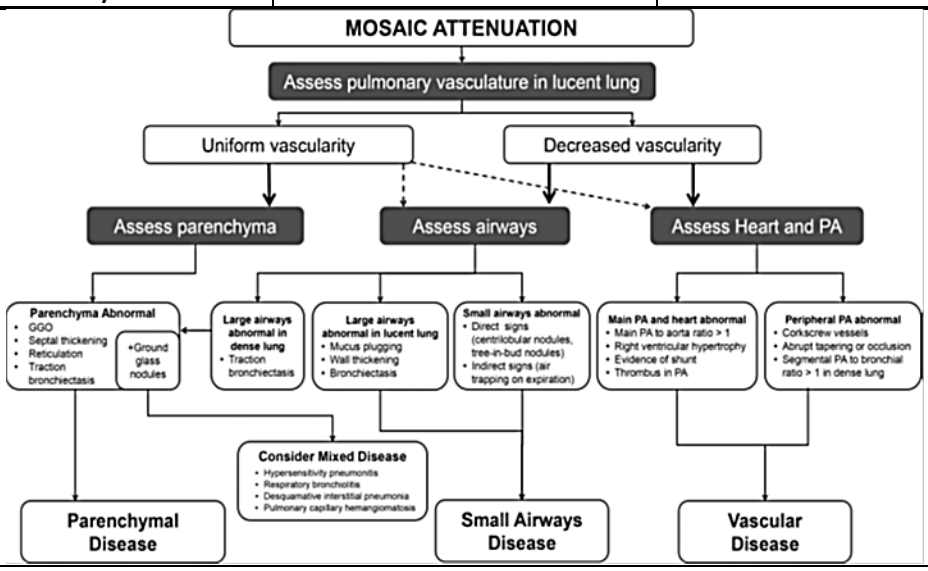
Mosaic Attenuation Pattern

Areas with increased attenuation adjacent to areas of decreased attenuation.

Differential diagnosis is based on:

- Caliber of the blood vessels
- Delineation of ground-glass opacity
- Density changes after deep expiration

Small Air-way disease (Bronchiolitis Obliterans)	Vascular disease (Chronic Pulmonary Embolism)	Parenchymal disease (GGO)
Mosaic Perfusion		Ground Glass Opacity
Low attenuation area is abnormal.		High attenuation areas are abnormal
Redistribution of the blood to the normal lung areas causes increased perfusion and increased attenuation		Low attenuation areas are normal
Reduced vessel size in low attenuation area		Normal vessel size
Borders between low & high density areas are sharp		ill defined
Pattern is accentuated in expiratory CT because of air-trapping in low density areas	No pattern accentuation on expiratory CT	No pattern accentuation on expiratory CT

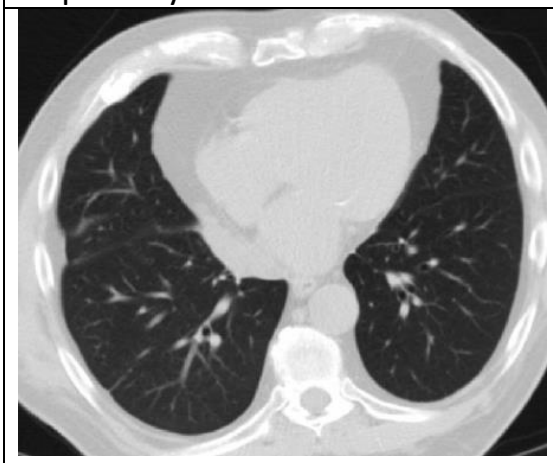
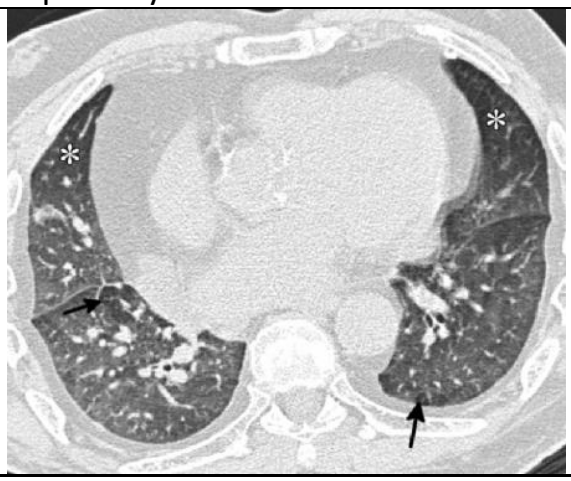


Mosaic attenuation due to normal physiologic gradient

Inspiratory CT => Normal lung attenuation

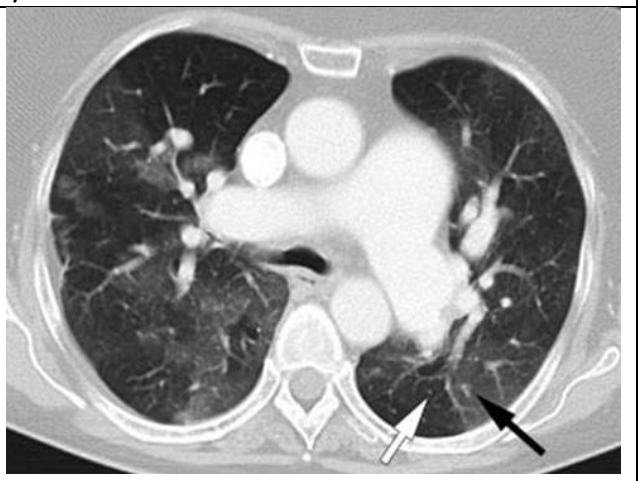
Expiratory CT => Mosaic attenuation due to:

- Posterior dependent lung areas: high attenuation caused by gravity-related increase in perfusion and decrease in intra-alveolar air
- Anterior areas of the lung (*): lower attenuation

Inspiratory CT	Expiratory CT
	

Mosaic perfusion secondary to chronic pulmonary embolism

Reduced vessel size in low attenuation area (white arrow) compared to high attenuation areas (Black arrow)



Mosaic attenuation pattern secondary to GGO

Inspiratory CT: Vessel calibre in low attenuation = high attenuation areas

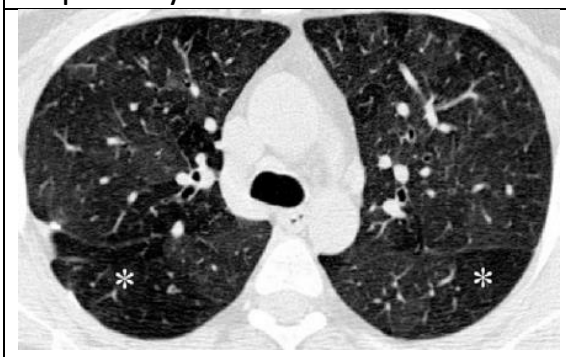
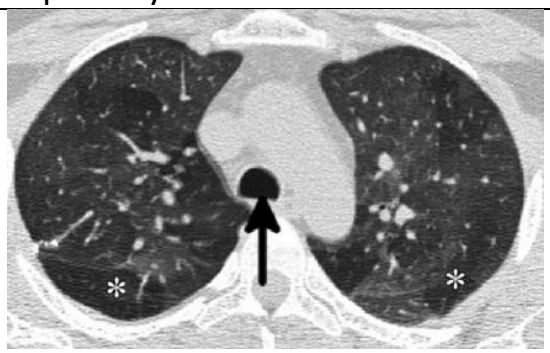
Expiratory CT: No pattern accentuation on expiratory CT

Inspiratory CT	Exiratory CT

Mosaic attenuation due to air trapping due to constrictive bronchiolitis

Inspiratory CT: Vessel caliber in low attenuation < high attenuation areas

Expiratory CT: Areas of hypoattenuation in inspiratory CT have not changed in attenuation in expiratory CT => difference in attenuation between the normal and abnormal areas becomes much more pronounced due to air trapping (*).

Inspiratory CT	Expiratory CT
	

Air Space Disease

Presentation:

A. Ground glass opacities + septal thickening
B. Consolidation + air-bronchogram
C. Centrilobular nodules

Causes:

A) Edema:

Cardiac pulmonary edema
ARDS

B) Blood:

Trauma – Pulmonary Contusion
Pulmonary Embolism – Pulmonary Infarction
Diffuse Alveolar Hemorrhage

C) Proteinaceous fluid:

Alveolar proteinosis

D) Inflammatory – Infection:

Bacterial – Viral – Fungal – TB

E) Inflammatory – Noninfections:

Allergic hypersensitivity pneumonitis (HP)
Cryptogenic Organizing Pneumonia (COP)
Alveolar sarcoid
Aspiration pneumonia
Lipoid pneumonia
Eosinophilic lung disease

F) Tumor cells:

Bronchoalveolar Carcinoma - Lymphoma

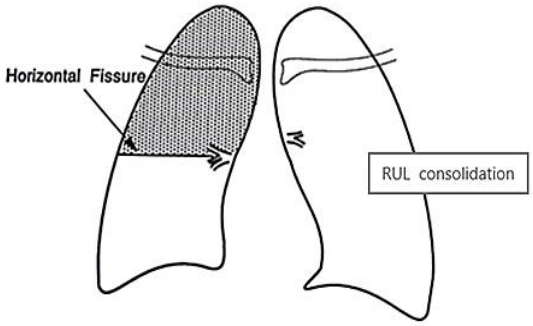
I- Pulmonary Infection

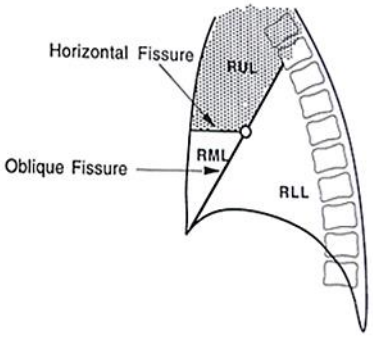
A- Pneumonia

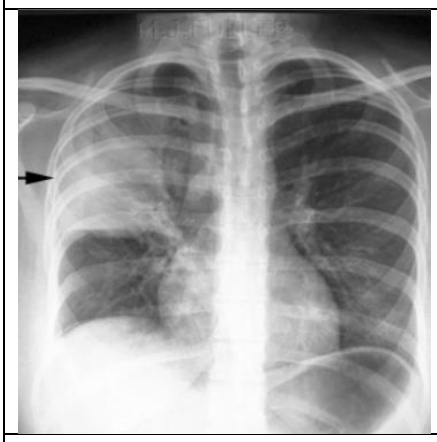
❖ Radiological Patterns:
1) Lobar pneumonia: Consolidation of a single lobe + Air-bronchogram. usually bacterial in origin (Strept. Pneumonia – Pneumococcal pneumonia) most common presentation of community acquired pneumonia.
2) Bronchopneumonia: Multifocal patchy consolidation usually involving several lobes Most commonly due to Staph. Aureus – Klebsiella – E.coli
3) Interstitial pneumonia caused by inflammatory cells located predominantly in the interstitial tissue of the alveolar septa Diffuse or patchy ground glass opacification. Caused by viral pneumonia, Mycoplasma, Chlamydia, or Pneumocystis.
4) Round pneumonia is an infectious mass-like opacity + air-bronchogram within Seen only in children Most commonly due to Streptococcus pneumoniae. Infection remains confined due to incomplete formation of pores of Kohn.
❖ Pathogens
A) Bacterial: Strept. Pneumoniae – Staph. – Pseudomonas – Klebsiella – Haemophilus influenza – Legionnella – Mycoplasma pneumoniae – Anaerobs – Actinomycosis - TB
B) Viral: Influenza – Varicella – Rubella – CMV
C) Fungal: Histoplasmosis – Candidiasis – Aspergillosis
D) Parasitic: Pneumocystis jiroveci (pneumocystis carinii)
❖ Risk Factors:
Alcoholic – Old age – Aspiration Chronic lung disease: Chronic bronchitis – Bronchiectasis – Cystic fibrosis Immunosuppression (Renal failure, DM, Cancer, Steroids, AIDS)

Lobar Pneumonia

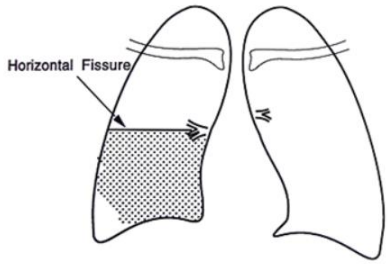
RUL Consolidation

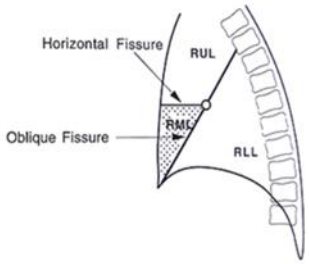


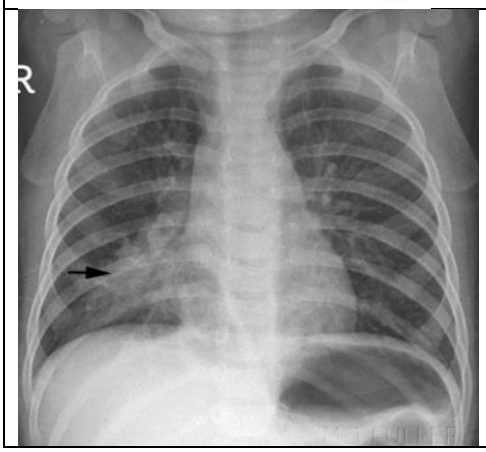


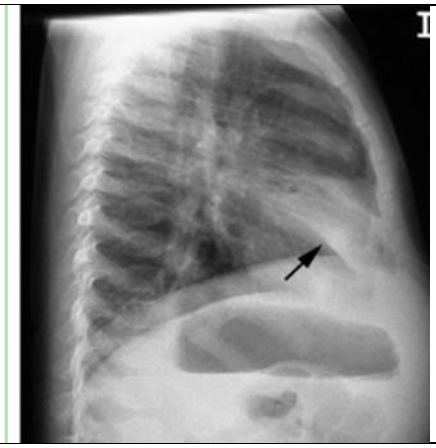


RML Consolidation









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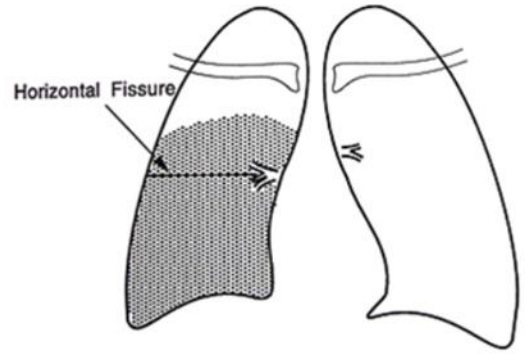

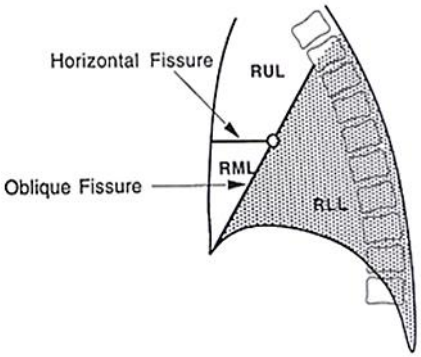

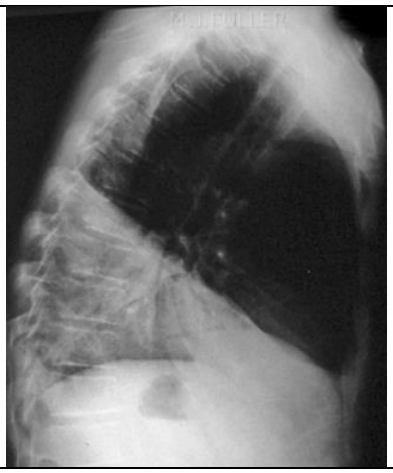

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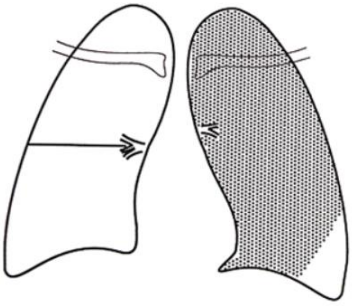

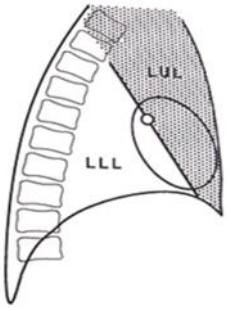

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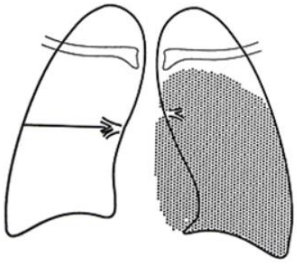
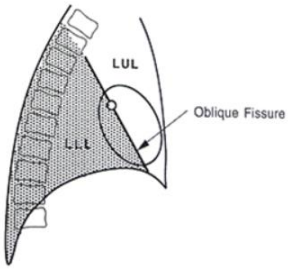
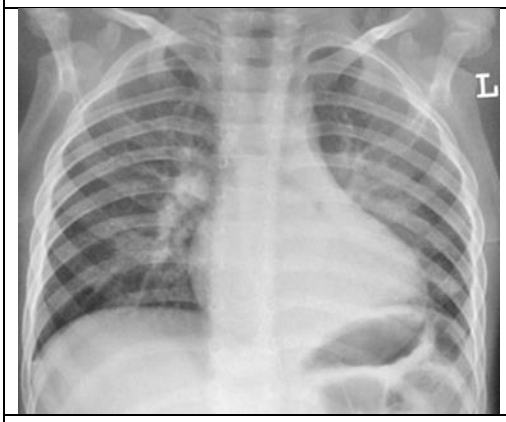
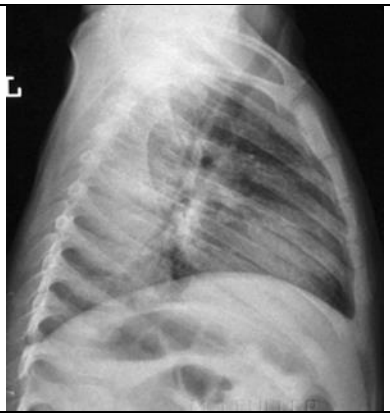
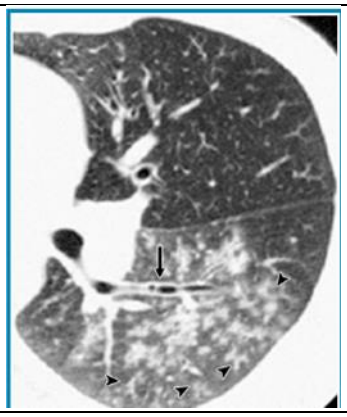




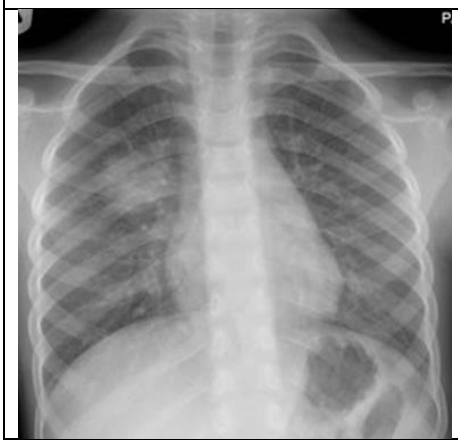


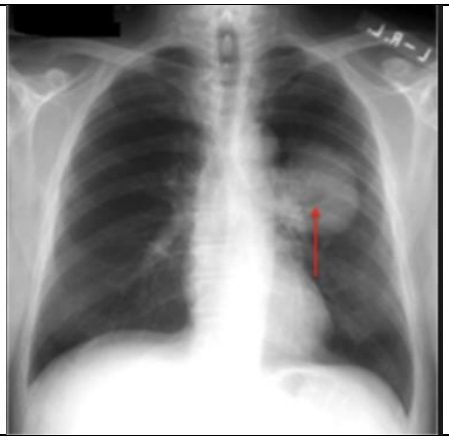


Bronchopneumonia



Round Pneumonia





❖ Complications of Pneumonia:

1) Pulmonary Abscess

is necrosis of the lung parenchyma
due to Staphylococcus aureus, Pseudomonas, or anaerobic bacteria.

Pulmonary cavity:

- Content: Air–fluid level
- Wall thickness: thin <4mm
- Inner wall: Smooth
- Surrounded by: Consolidation

2) Pulmonary Gangrene

very rare complication of pneumonia
Extensive necrosis or sloughing of a pulmonary segment or lobe.
is a severe manifestation of pulmonary abscess.

3) Pneumatocele

is a thin-walled, air-filled thin walled cyst
post-traumatic or develop as a sequela of pneumonia
typically from Staphylococcus aureus or Pneumocystis.
Pneumatoceles almost always resolve

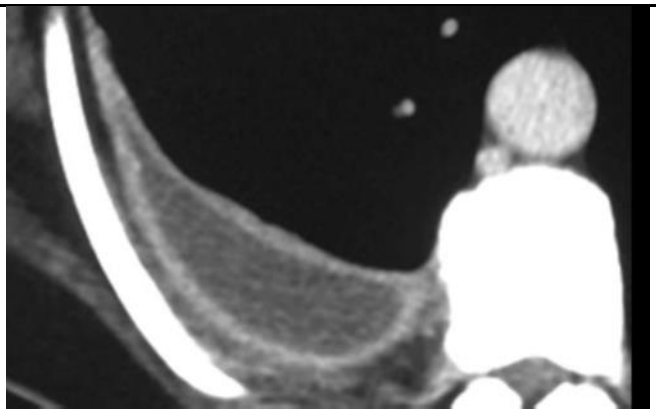
4) Parapneumonic effusion:

Stage 1: Exudation = free fluid
Stage 2: Fibropurulent
Stage 3: Organization, erosion into lung or chest wall

4) Empyema

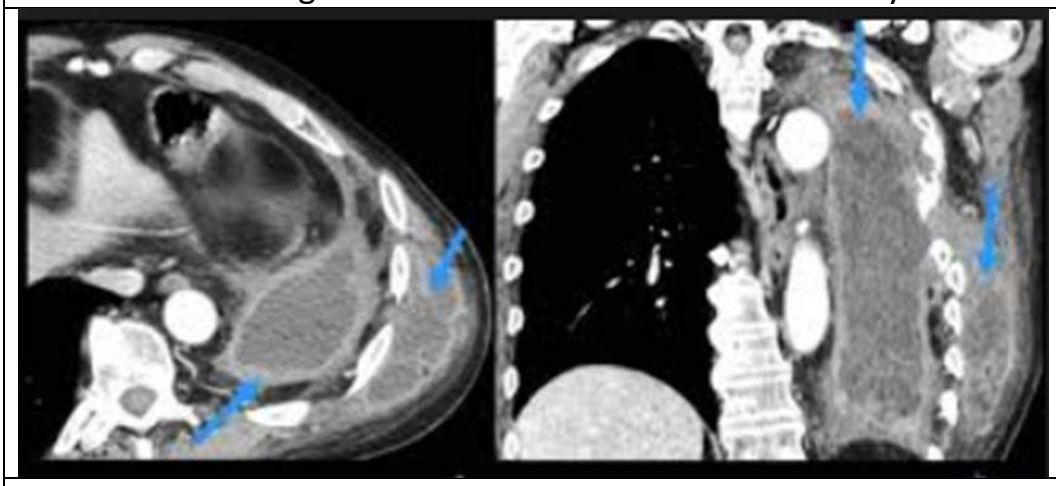
Empyema is infection within the pleural space.

Split pleura sign = Enhancing parietal and visceral pleural surfaces + separated by pleural fluid



5) Empyema Necessitans

is extension of an empyema to the chest wall
Most commonly secondary to tuberculosis.
Other causative organisms include Nocardia and Actinomyces.



6) Bronchopleural Fistula (BPF):

is an abnormal communication between the airway and the pleural space.
the most common cause of BPF is surgery
other etiologies include lung abscess, empyema, and trauma.
On imaging, new or increasing air is present in a pleural effusion.
A connection between the bronchial tree and the pleura is not always apparent.

❖ Resolution of Pneumonia

80-90% => Resolve within 4w
5-10% => Resolve within 4-8w
Non-clearance:

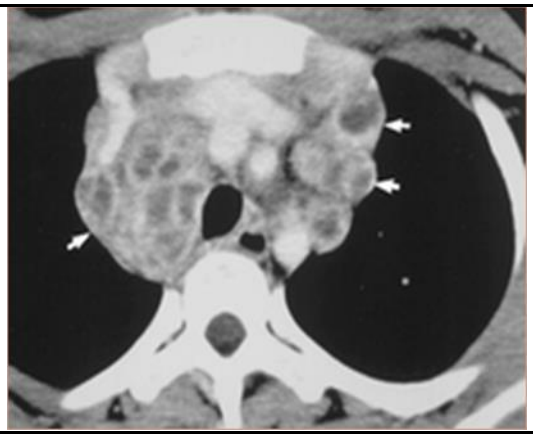
- Antibiotic resistance
- Consider other pathogen (eg: TB)
- Obstruction pneumonitis due to tumor

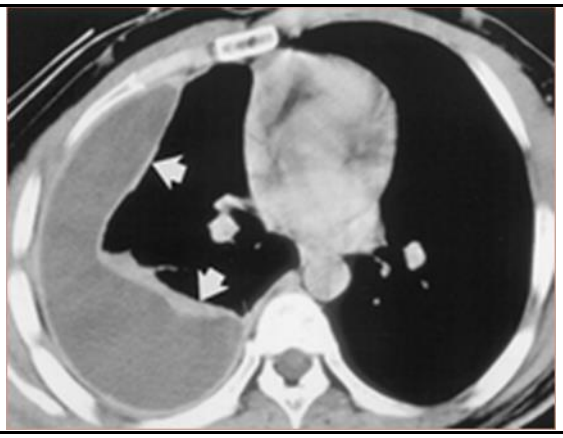
B- Pulmonary Tuberculosis (TB)

- ❖ Causative Organism: Mycobacterium tuberculosis
- ❖ Mode of transmition: Inhalation of infected droplets
- ❖ Risk factors:
  Low socioeconomic scale (Homeless) – Alcoholics – Immigrants – Prisoners - Elderly patients - AIDS

A) Primary TB => Ranke complex

- Upper segment of lower lobe > Lower part of upper lobe
- Gohn focus = ill-defined consolidation: Lower lobes > Upper lobes
- Pleural effusion
- Lymphadenopathy: Hilar – paratracheal LNs
  → Size >2cm + Central low attenuation + Peripheral enhancement
- Cavitation is rare.





B) Post – primary TB:

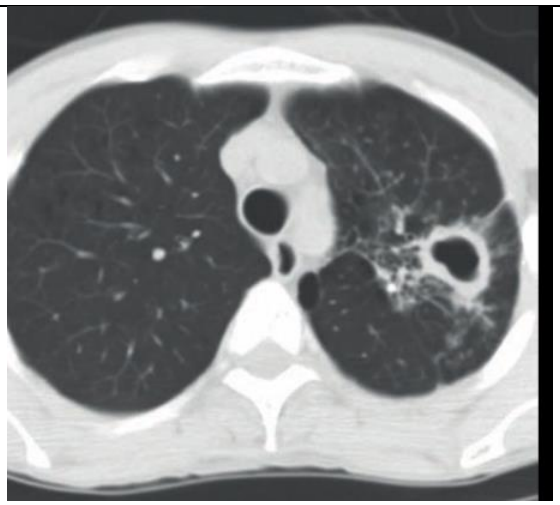
- Caused by reactivation of dormant infection acquired earlier in life

Pattern:

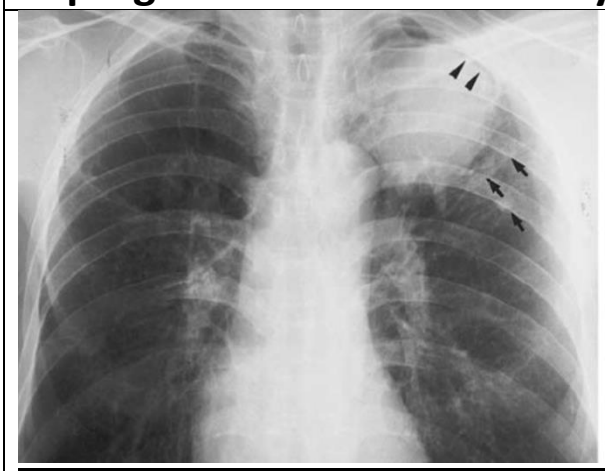
- Tree-in-bud nodules: suggest active endobronchial spread.
- Exudative pattern: Patchy or confluent consolidation
- Fibro-productive:
  - Consolidation => TB Cavity ± Fungal ball (Aspergilloma)
  - Reticulo-nodular infiltration.
  - Apical fibrosis
    - ⇨ Architecture distortion + Volume loss
    - ⇨ Apical pleural thickening
    - ⇨ Hilar retraction + Mediastinal shift
    - ⇨ Traction bronchiectasis + Paracicatricial emphysema

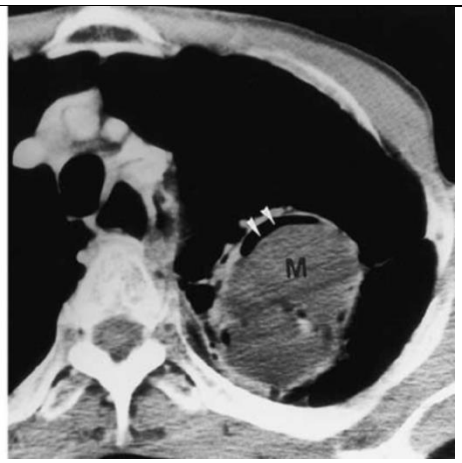
- Distribution: Upper lung zones = Apical and Posterior segments of both UL + Apical segment of LL
- No significant mediastinal or hilar LNs

Apical TB cavity

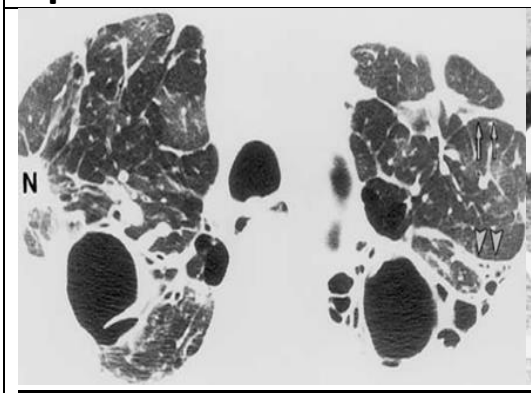


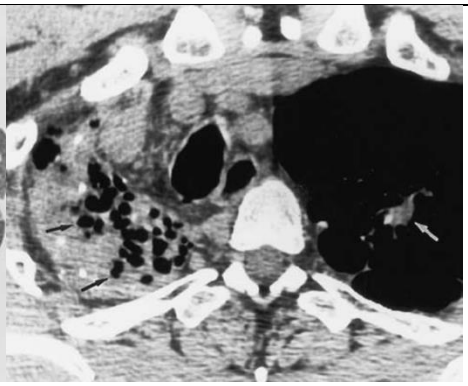
Aspergilloma within TB Cavity





Apical Fibrosis





Healed TB in Good Immunity :

- ⇨ Healing without residual
- ⇨ Apical scarring
- ⇨ Calcified granuloma (Tubercloma)
- ⇨ Calcified LN (Broncholithiasis)

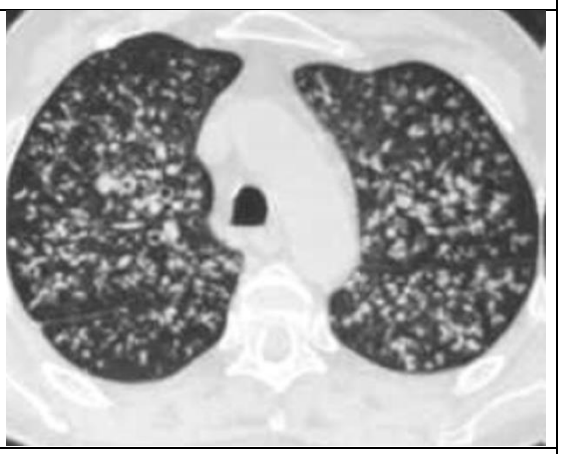
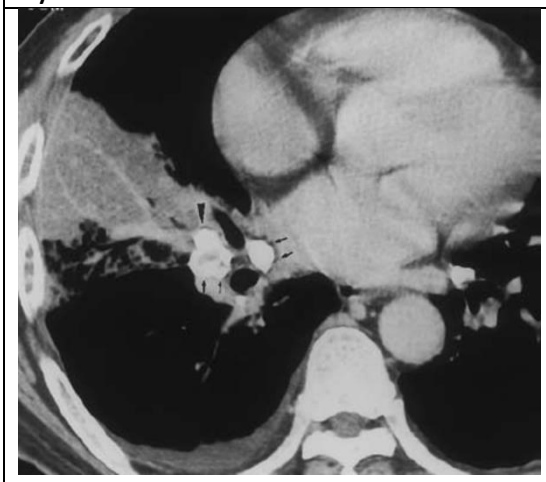
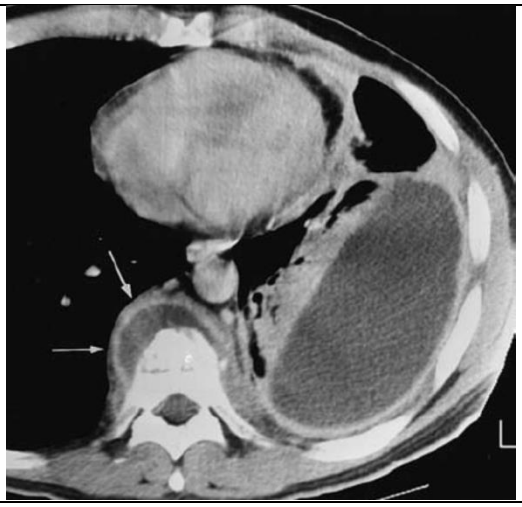
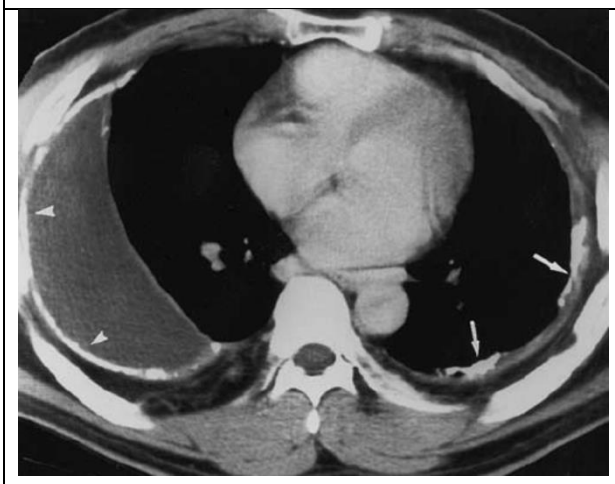
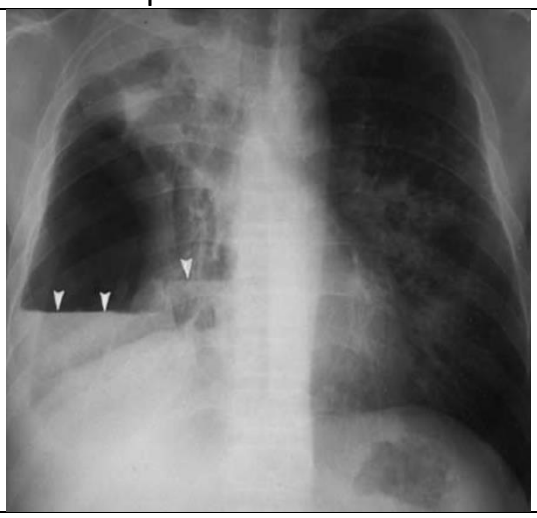
Complication of TB:

Complications of LNs => Rupture into:

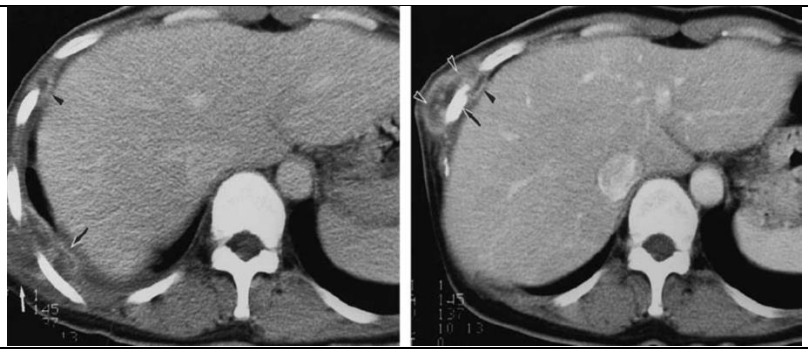
- Pericardium => Constrictive pericarditis = Pericardial thickening (>4mm) + Calcification.
- Bronchi => bronchilthiasis => Collapse - Hyperinflation
- Trachea => Tracheal traction diverticulum
- Esophagus => Esophagomediastinal fistula.

Complications of TB cavity => Rupture into:

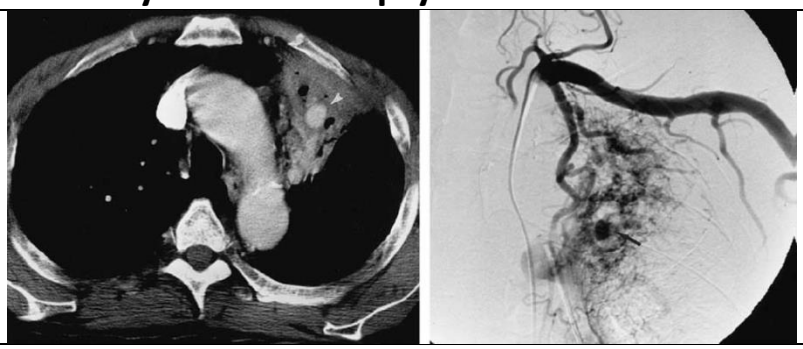
- Pleura:
  - → Pneumothorax
  - → Empyema: Split pleural sign
  - → Fibrothorax: Calcifications of the parietal and visceral pleura
  - → Bronchopleural Fistula (BPF)
- Chest wall => Empyema Necessitatis = Empyema + Chest wall subcutaneous abscess
- Spine => Pott disease = Discitis + Vertebral endplates irregularity and sclerosis + Vertebral wedging & Kyphosis + Paravertebral abscess
- Airway => Bronchogenic spread => Miliary TB
- Pulmonary vessel =>
  - → Rasmussen aneurysm => Hemoptysis
  - → Vascular spread => CNS:
    - Basal leptominengeal nodular thickening + enhancement.
    - Tubercloma in basal ganglia + Nodular enhancement

Miliary TB	
	
Broncholithiasis => Middle lobe Syndrome	TB Spondylitis
	
Fibrothorax	Broncho-pleural Fistula
	

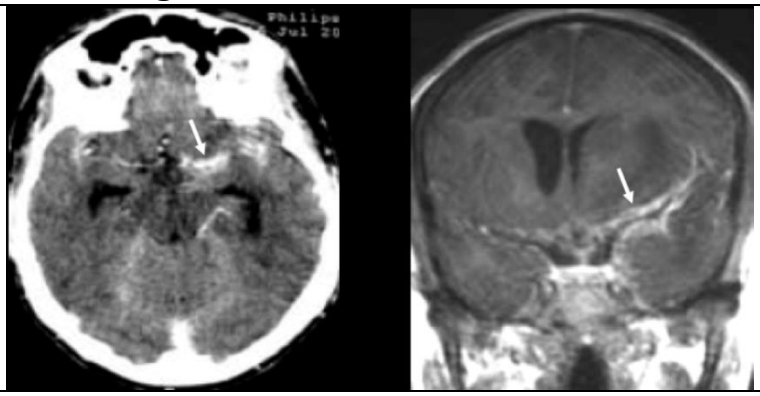
Empyema Necessitatis



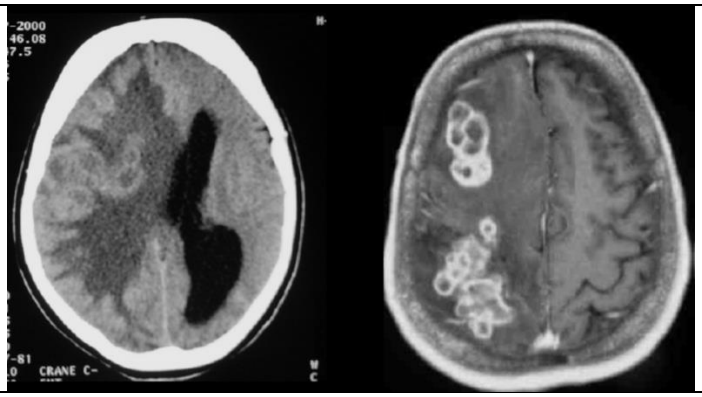
Rasmussen aneurysm => Hemoptysis



CNS TB: Basal menegeal nodular enhancement



CNS TB Granuloma + Nodular enhancement



NONTUBERCULOUS MYCOBACTERIAL INFECTION (NTMB)

ATYPICAL MYCOBACTERIAL INFECTION

Pathogen: Mycobacterium avium intracellulare (MAI) and M. kansasii

Mode of transmition:

Not acquired by human – human transmission

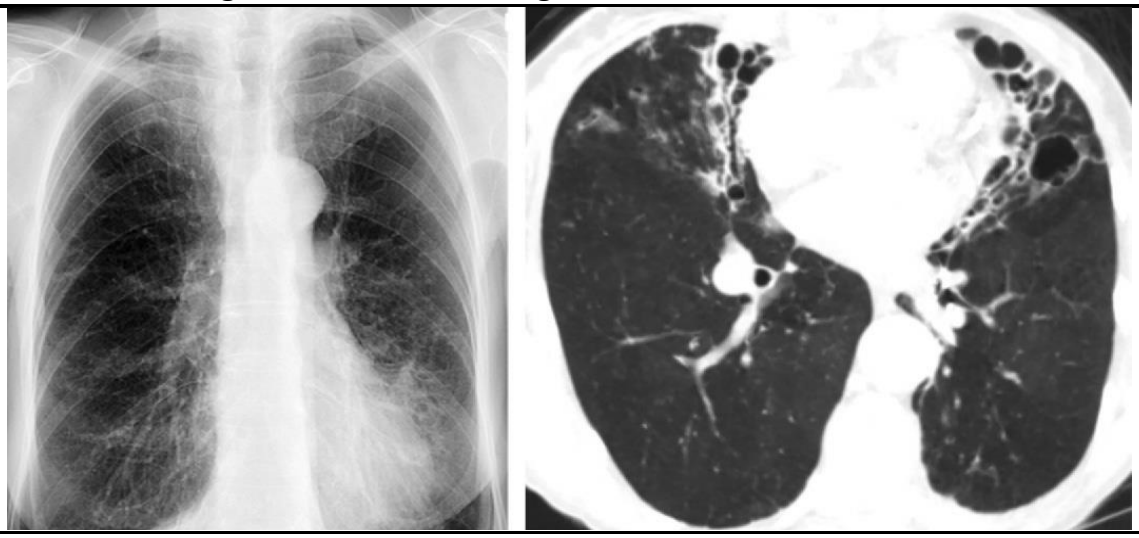
But direct infection from soil or water

- **Older females in good health with cough, low-grade fever, and weight loss, called Lady Windermere syndrome**
- **Older men with COPD**
- **AIDS patients**

Classic radiographic findings

Pattern: Bronchiectasis and tree-in-bud nodules

Distribution: Right middle lobe – Lingula.



C- Fungal Infection

Endemic Mycoses	Opportunistic Mycoses
Prevalent only in certain geographical areas	Worldwide distribution In immunocompromised patients
1) Histoplasmosis 2) Coccidiomycosis 3) Blastomycosis	1) Aspergillosis 2) Candidiasis 3) Cryptococcosis 4) Mucormycosis

Histoplasmosis

Pathogen: Histoplasma capsulatum
Geographical area: Ohio – Mississippi – St. Lawrence river valleys in soil contaminated with bat or bird guano.
Radiological features: **A) Consolidation (Primary histoplasmosis)** Parenchymal consolidation + Adenopathy **B) Nodular form (Chronic histoplasmosis)** Histoplasmoma = Solitary well defined calcified nodule (Granuloma) Fibrocavitary = Post-primary TB **C) Disseminated form (Immunocompromised patients)** Miliary nodules Calcifications in liver and spleen **E) Mediastinal Histoplasmosis** Calcified mediastinal and hilar LNS (Broncholithiasis) => Obstructive atelectasis Mediastinal fibrosis (Fibrosing mediastinitis) => SVC syndrome

Aspergillosis

Immune status	Disease
Hypersensitivity	**ABPA**
Normal	**Aspergilloma**
Mild suppression	**Semi-invasive**
Severe suppression	**Airway Invasive - Angioinvasive**

A) Allergic Bronchopulmonary Aspergillosis (ABPA)

hypersensitivity reaction to aspergillus
Most commonly in patients with long-standing asthma.

Radiological Findings:

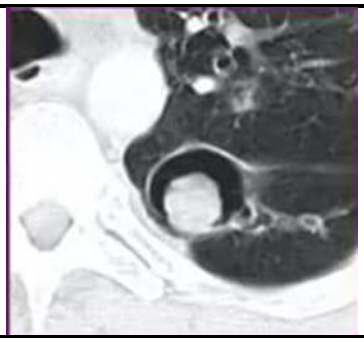
Upper lobe
Saccular bronchiectasis
Mucoid impaction
⇨ **Finger in glove**

B) Aspergilloma = Mycetoma = Fungal Ball

Saprophytic infection in a preexisting pulmonary cavity
Causes:
TB – End stage sarcoid – PMF of silicosis – Emphysematous bulla
Congenital anomalies: Bronchogenic cyst - Sequestration
Post-infectious/post-traumatic pneumatocele.

Radiological Features:

UL cavity
Dependent sign = Fungal ball moves with position
Monod sign = curvilinear air surrounding the aspergilloma.



C) Semi-Invasive Aspergillosis

is a necrotizing granulomatous inflammation (analogous in pathology to reactivation TB) in response to chronic aspergillus infection.

Mild immunocompromised patients:

- Debilitated, Diabetic, Alcoholic, and COPD patients

Progress slowly over months or years.
Mortality = 30%

Radiological Features:

- Segmental areas of consolidation
- Cavitation (6m after infection)

D) Invasive Aspergillosis:

Marked immunocompromised patients: Neutropenic - AIDS patients

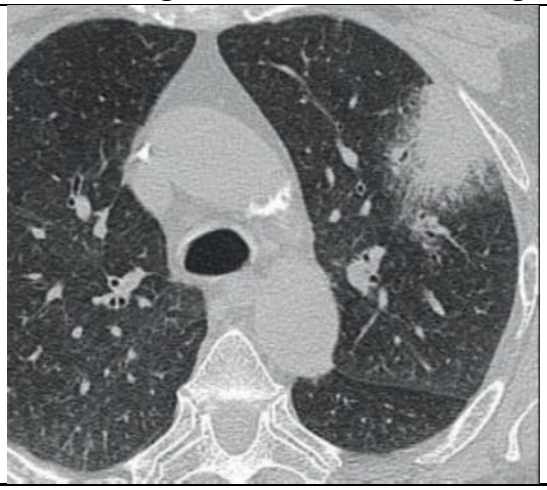
High mortality rate = 70-90%

Airway-invasive Aspergillosis:

Centrilobular and tree-in-bud ground glass nodules.

Angio-invasive Aspergillosis:

CT halo sign = halo of ground glass attenuation surrounding consolidation.
The ground glass corresponds to hemorrhagic infarction of the lung.



Recovery phase:

Air crescent sign = The nodules cavitate
Non-dependent nodule

D- Thoracic Manifestations of AIDS

AIDS = Acquired Immunodeficiency Syndrome

HIV = Human Immunodeficiency Virus

Thoracic Manifestations in 60% of patients of AIDS:

A) Noninfectious airway disorders

- Bronchial abnormalities:
  - Bronchial wall thickening
  - Bronchiectasis
  - Bronchiolitis: Ground glass centrilobular nodules + Tree in bud
- Ventilation abnormalities (Small airway disease)

B) Chronic inflammatory conditions

- Granulomatosis (= Sarcoid like):
  - Perilymphatic Nodules / Reticulonodular
  - Mediastinal lymphadenopahty
  - Pulmonary Fibrosis
- Interstitial pneumonias (LIP – UIP – NSIP):
  - LIP: GGO + Septal thickening + perivascular cysts.
  - UIP = IPF: Septal thickening + Traction brochiectasis + Honey combing (Peripheral & Basal)
  - NSIP: GGO + Septal thickening

C) Opportunistic Infections:

- Pneumocystis Jiroveci Pneumonia
- Mycobacterial infection (TB – MAI)
- Bacterial pneumonia (S.pneumoniae – Haemophilus influenzae)
- Fungal infection
- CMV pneumonia

D) Benign Lymphoproliferative Disorders:

- Parenchymal lymphoid hyperplasia => Multiple small nodules
- Reactive follicular hyperplasia => Regional lymphadenopathy

D) Tumors in AIDS:

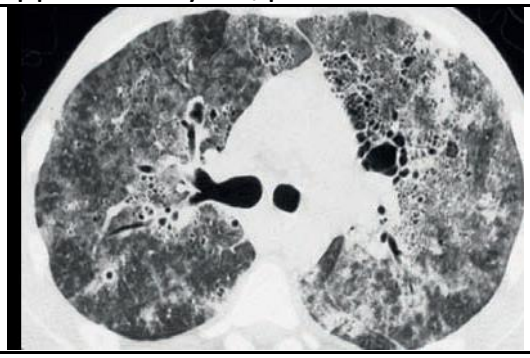
- Kaposi sarcoma
- Lymphoma
- Thymic tumors
  - Good Syndrome = Thymoma (Anterior mediastinal mass) + HIV

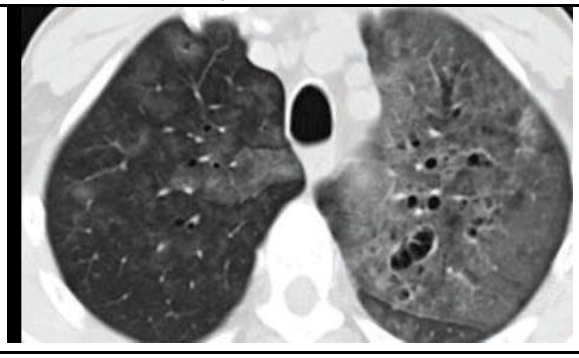
Opportunistic Fungal Infection:

Pneumocystis Jiroveci Pneumonia = Pneumocystis Carinii Pneumonia

Opportunistic fungus in individuals with CD4 counts <200 cells/cc.

Bilateral perihilar (central) GGO + Centrilobular nodules
Crazy paving = GGO + Septal thickening
Upper lobe cysts /pneumatocele => Pneumothorax /Pneumomediastinum.





Cryptococcosis

Pathogen: Cryptococcus Neoformans
is the most common fungal infection in AIDS patients.
90% CNS Involvement: Pulmonary infection coexists with meningitis.
CD4 count is less than 100 cells/cc in affected individuals.

Ground glass attenuation - Focal consolidation - Cavitating nodules.
Miliary disease - Lymphadenopathy – Effusion

Kaposi Sarcoma

KS is the most common AIDS-related neoplasm worldwide.

- Homosexual men.
- Consolidation perihilar peribronchovascular
- Ill defined pulmonary nodules.
- Interlobular septal thickening + GGO.
- Pleural effusions
- Mediastinal LNs
- Nuclear Medicine: KS is not gallium-avid (DD: Lymphoma and Infection)

II- Pulmonary Edema

Aetiology:

↑ Hydrostatic pressure: Cardiogenic - Renal

↓ Oncotic pressure: Hypoproteinemia (Cirrhosis)

↑Capillary permeability: ARDS

Lymphatic obstruction: Mediastinal tumors

A- Cardiogenic Pulmonary Edema

Aetiology:

i. Adults:
   a. LVF from coronary artery disease
   b. Valvular disease: MS / MR
   c. Lt atrial thrombus / Myxoma

ii. Neonates:
   a. TAPVR
   b. Hypoplastic left sided heart
   c. Cor triatrtum

Grading:

Grade 1 = Cephalization = Redistribution:

Pulmonary capillary wedge pressure (PCWP) = 10 -17 mmHg

- Moustache sign = Diameter of UL veins ≥ LL veins at comparable distance from hilum

(Normally: Diameter of UL veins ½ diameter of LL veins)

Grade 2 = Interstitial Edema:

Pulmonary capillary wedge pressure (PCWP) = 18 – 25 mmHg

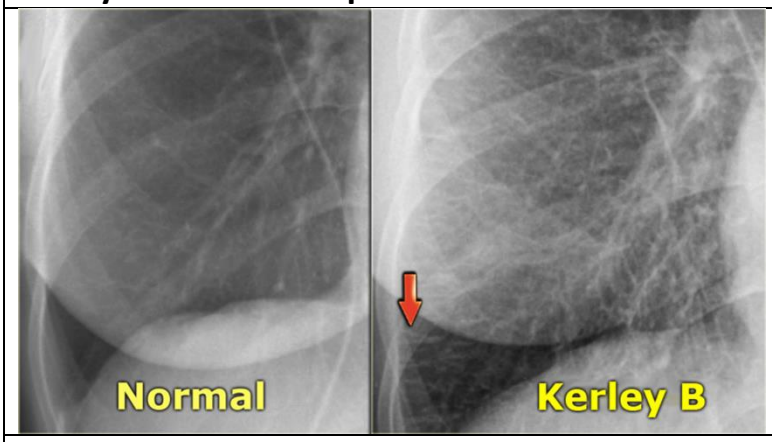
- Karley's A lines = Central = Peribronchovascular thickening / cuffing
- Karley's B lines = Peripheral = Interlobular smooth septal thickening
- Perihilar haziness
- Lamellar effusion = shallow collection between visceral pleura and lung surface which sometimes spares costophrenic angles

Grade 3 = Alveolar Edema:

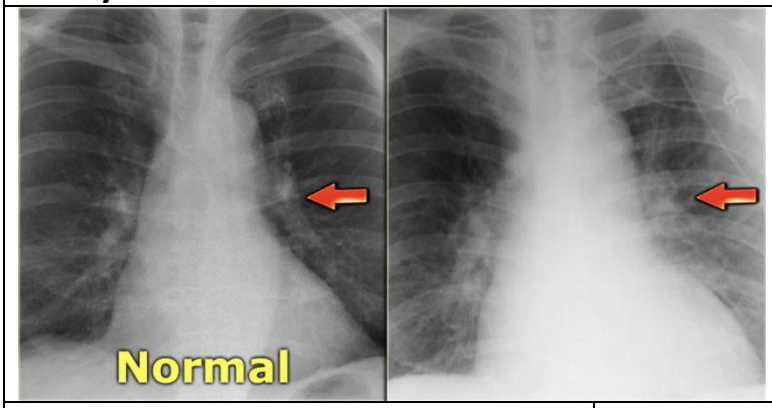
Pulmonary capillary wedge pressure > 25 mmHg

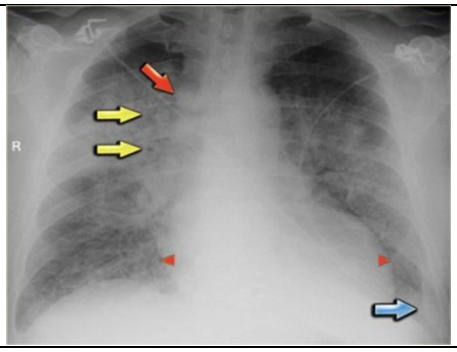
- Batwing sign = Bilateral central perihilar patchy consolidation / GGO
- Pleural effusion

Karley's B lines = Peripheral = Interlobular smooth septal thickening



Kerley's A lines = Perihilar haziness



Interstitial edema = Smooth septal thickening	Alveolar edema + Pleural effusion
	

Increased diameters of the superior pulmonary vessels

B- Acute Respiratory Distress Syndrome (ARDS)

Non-Cardiogenic Pulmonary Edema

Aetiology:

Pulmonary causes:	Extra-pulmonary causes
Lung contusion Diffuse pulmonary infection Smoke / Chlorine gas / Nitrogen dioxide inhalation Near drowning Massive aspiration of gastric contents (Mendelssohn's Syndrome) Fat or amniotic fluid embolism	Severe trauma Cardiopulmonary bypass Hypertransfusion Sepsis Burns Acute pancreatitis Disseminated intravascular coagulation (DIC) Heroin/methadone/crack cocaine overdose

Pathophysiology:

Diffuse lung injury => increase capillary permeability => Flooding of alveolar spaces with exudate

Clinical picture:

Acute onset of dyspnea and hypoxemia

Acute phase = Exudative phase (1-7 days):

- Pattern: Consolidation / GGO
- Distribution: Bilateral patchy / multifocal / diffuse
- Gravity dependent
- **No cardiomegaly**
- **No pleural effusions**
- **No Kerley A&B lines**

Intermediate Phase (8–14 Days)

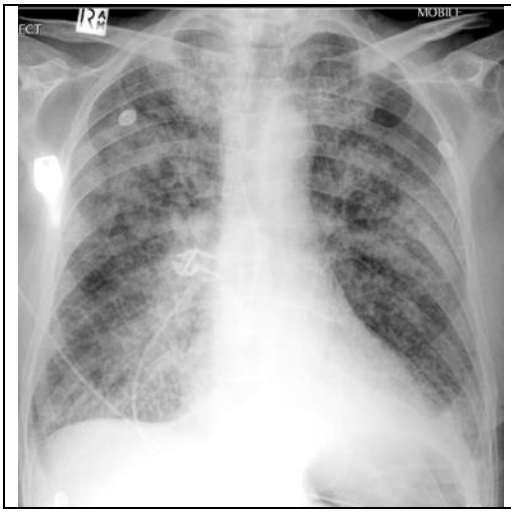
Radiographic appearances usually stabilize and remain static

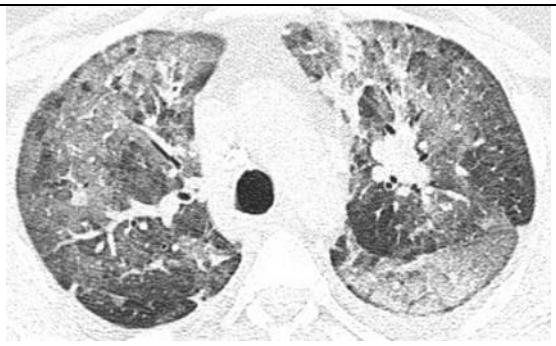
Late phase = Fibrotic phase (>15 days)

Most radiographic pattern Resolve

- **Pattern:** Reticular interstitial disease => Fibrosis => Traction bronchiectasis

- **Distribution:** Anterior distribution (right middle lobe and lingula)
- **Mechanism:**
  - Parenchymal damage intrinsic to ARDS
  - Barotrauma resulting from prolonged mechanical ventilation
  - The dependent portions of the lungs are protected from barotrauma by typical gravitational gradient of consolidation in ARDS





	Cardiac	Renal	Injury
Heart size	Enlarged	Enlarged	Not enlarged
Vascular pedicle	Normal or enlarged	Enlarged	Normal or reduced
Pulmonary blood flow distribution	Inverted	Balanced	Normal or balanced
Pulmonary blood volume	Normal or increased	Increased	Normal
Septal lines	Not common	Not common	Absent
Peribronchial cuffs	Very common	Very common	Not common
Air bronchogram	Not common	Not common	Very common
Lung edema, regional distribution (horizontal axis)	Even	Central	Peripheral
Pleural effusions	Very common	Very common	Not common

III- Diffuse Pulmonary Hemorrhage

Diffuse Alveolar Hemorrhage (DAH)

Immune mediated Pulmonary Vasculitides Pulmonary Renal Syndrome	Non-immune mediated
Small vessel vasculitis (ANCA +ve) Wegner's granulomatosis Microscopic polyangiitis Churg-Strauss Syndrome **Immune mediated vasculitis:** Goodpasture's syndrome Henoch-Schonlein Purpura **Collagen vascular disease:** SLE – RA Polymyositis – Dermatomyositis Scleroderma Antiphospholipid Abs **Secondary:** Drug induced Inflammatory bowel disease Paraneoplastic Transplantation	Idiopathic pulmonary hemosiderosis Drugs: Penicillin Coagulopathies Mitral stenosis

Radiological features:

Pulmonary Renal Syndrome = DAH + Glomerulonephritis (GN)

Pattern: Diffuse alveolar hemorrhage (DAH)

- Air space opacities = Consolidation + GGO
- Crazy paving pattern = GGO + septal thickening

Distribution:

- Bilateral & Perihilar (Batwing)
- Middle & lower lung zones
- Sparing lung apex and costophrenic angles

Repeated attacks of hemorrhage => Hemosiderin deposition => Reticular pattern => Pulmonary fibrosis

1) Wegener Granulomatosis (WG)

Systemic necrotizing non-caseating granulomatous small-vessel vasculitis
Classic clinical triad = Sinusitis + lung involvement + Renal insufficiency
C-ANCA is positive.

Upper Respiratory Tract:

Nasal ulceration
Sinusitis: granulomatous masses + bone destruction
Tracheal circumferential nodular wall thickening + No calcification + Posterior wall involvement

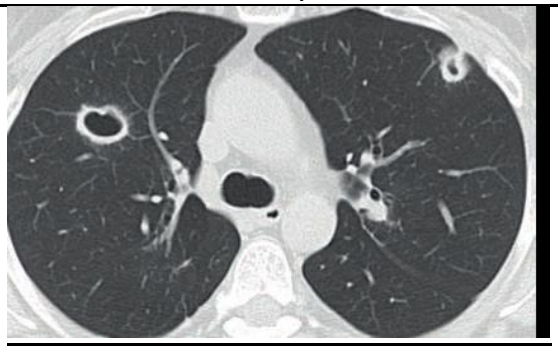
Pulmonary radiological features:

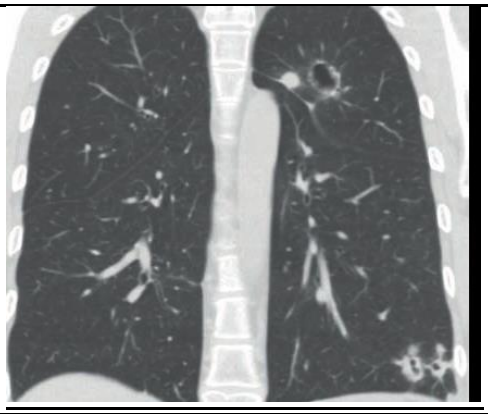
A) Waxing and waning pulmonary nodules + masses (70%):

- Random distribution: mainly peripheral subpleural
- CT halo sign= ground-glass opacities around mass
- Radiating linear scar, spiculations, Pleural tags
- Cavitary pulmonary nodules: Thick wall + irregular inner margin

B) Ground glass opacities and consolidation (50%):

- Pulmonary hemorrhage.
- Peripheral wedge shaped
- Perihilarl and peri-bronchovascular.





2) Microscopic Polyangiitis

Most common cause of Pulmonary-Renal Syndrome
Pulmonary hemorrhage (DAH) + Renal disease (Glomerulonephritis).
P-ANCA is positive.

3) Goodpasture Syndrome:

Pulmonary hemorrhage (DAH) + Renal disease (Glomerulonephritis)
Antiglomerular basement membrane Abs

4) Churg-Strauss Syndrome = Allergic Angitis & Granulomatosis:

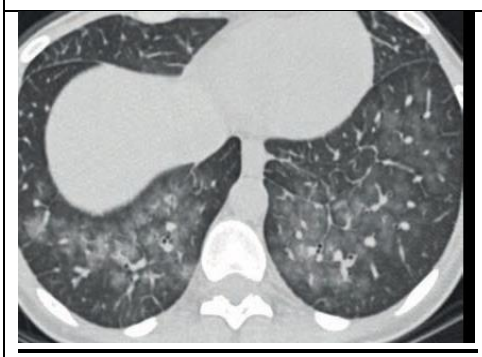
Pulmonary vasculitis + Asthma + Peripheral eosinophilia
Peripheral consolidation + Centrilobular nodules (non-cavitary)
Fleeting / transient opacities

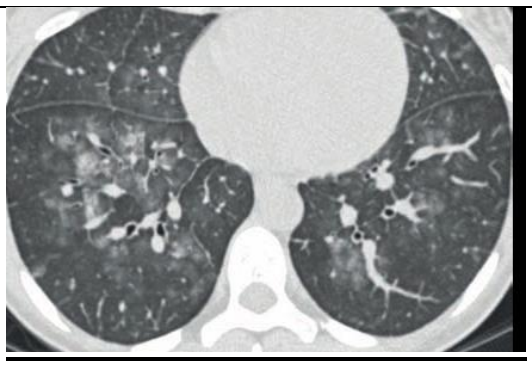
5) Henoch-Schonlein Purpura (IgA):

Young Boys.
Purpura + Arthritis + Abdominal pain (GIT Bleeding) + Kidneys

6) SLE:

Pleural disease: Thickening & effusion (Most common as RA)
Acute lupus pneumonitis = Acute interstitial pneumonia (AIP): Bi LL Consolidation & GGO
Diffuse alveolar hemorrhage (DAH): Bi central perihilar consolidation / GGO
DVT + PE
Shrinking lung syndrome: progressive loss of lung volume





IV- Eosinophilic Lung Disease

Eosinophilic lung disease is a spectrum of diseases that feature accumulation of eosinophils in the pulmonary airspaces and interstitium.

A- Idiopathic:

- Acute eosinophilic pneumonia (AEP)
- Chronic eosinophilic pneumonia (CEP)
- Simple pulmonary eosinophilia (SPE) (Loeffler's Syndrome)
- Idiopathic hypereosinophilic syndrome (HIS)

B- Secondary:

- Allergic Bronchopulmonary Aspergillosis (ABPA)
- Bronchocentric granulomatosis (BG)
- Helminth infection: Ascaris – Schistomiasis
- Drugs

III) Eosinophilic vasculitis:

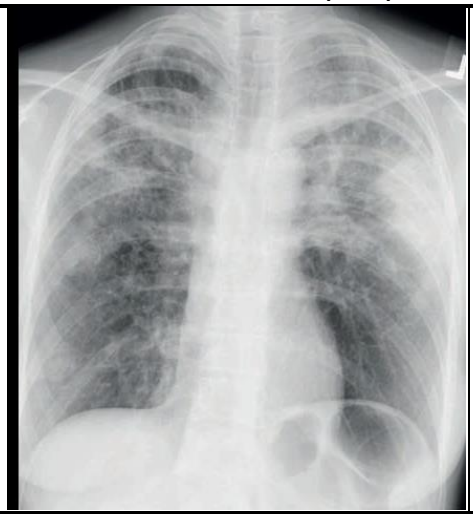
- Churg-Strauss Syndrome

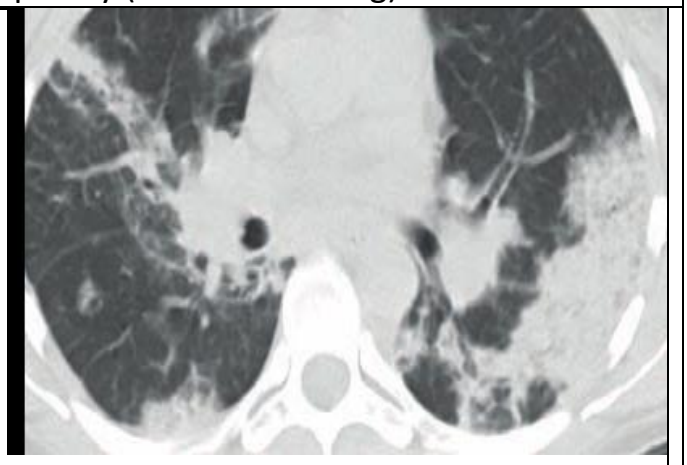
Diagnosis => One of the following:

a. Pulmonary opacities with peripheral eosinophilia
b. Tissue eosinophilia confirmed at open or transbronchial lung biopsy
c. Increased eosinophils in bronchoalveolar lavage (BAL) fluid

Radiological features:

Pattern: Airspace opacities = Consolidation + GGO + Centrilobular nodules
Distribution: Bilateral peripheral patchy (Reverse batwing)





	SPE	AEP	CEP	IHS
Asthma	No	NO	YES	NO
Peripheral Eosinophili	Yes	NO	**Peripheral Eosinophilia**	
Ig E Level	**Increased Ig E Level**			
Extra thoracic	No			**YES**
HRCT	Transient Migratory	Diffuse alveolar or mixed alveolar-interstitial opacities (as ARDS). Fever, dyspnoea and hypoxia Rapid improvement with steroids.	Homogenous peripheral consolidation	Diagnostic criteria: 1) Persistent eosinophilia > 6 months or death within 6 months 2) Absence of parasitic, allergic, or other known causes of eosinophilia 3) Evidence of organ involvement and multiorgan system dysfunction (Heart and CNS)

ABPA	BG	Parasitic	Drug	CSS
Asthma		No Asthma		Asthma
Peripheral Eosinophilia				
Increased Ig E Level				
No Extra-thoracic manifestations				
UL & central Bronchiectasis + Mucoid impaction => Finger in glove	Focal mass or lobular consolidation with atelectasis	Variable depending on type of parasitic infestation	Peripheral airspace consolidation and GGO	Peripheral airspace consolidation with lobular distribution. Fleeting opacities Centrilobular nodules

V- Pulmonary Alveolar Proteinosis (PAP)

A disease causing filling of the alveoli with a proteinaceous lipid-rich material (surfactant-like material).

3 Subtypes:

- Idiopathic PAP (90%)
- Secondary PAP (5-10%): Drug induced – Industrial inhalation – Immune compromise
- Congenital PAP (Rare, 2%)

Diagnosis:

Lung biopsy or BAL: intraalveolar deposits of proteinaceous material, dissolved cholesterol, and eosinophilic globules

Clinical picture:

Extensive sputum production (litres/day)

Radiological features:

Pattern**: Crazy paving appearance:**

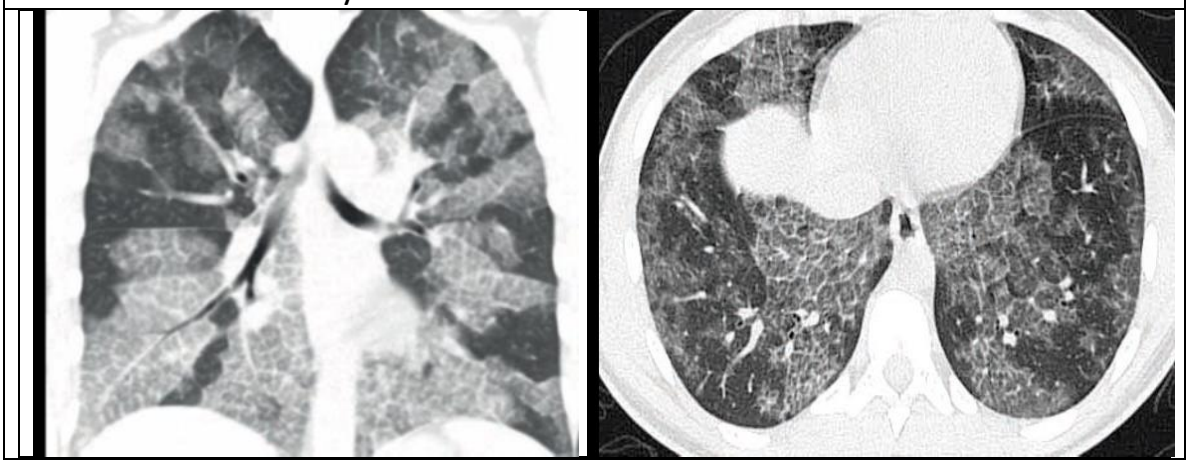
Geographical / patchy areas

GGO + smooth interlobular septal thickening + No air-bronchogram

Distribution: Central & perihilar

DD: Pulmonary edema

- ✓ No cardiomegaly
- ✓ No pleural effusion
- ✓ No Karley's A & B lines



VI- Hypersensitivity Pneumonitis (HP)

Extrinsic Allergic Alveolitis (EAA)

Hypersensitivity reaction to inhaled environmental antigens

Diagnosis:
Fluid from BAL usually demonstrates an increased number of white cells.
Lymphocytes accounting for at least 20%–30% of WBCs, commonly > 50%.

Farmer's lung – Bird fancier's ling

Examples of Hypersensitivity Pneumonitis

Disease	Antigen Source	Putative Antigen
Bird fancier's disease	Various birds	Protein in avian feces, feathers
Cheese worker's lung	Moldy cheese	*Penicillium* species
Coffee worker's lung	Coffee bean	Unknown
Farmer's lung	Moldy hay	Thermophilic actinomycetes
Furrier's lung	Animal fur	Protein in animal fur
Hot tub lung	Warm water	*Mycobacterium avium* complex
Humidifier lung	Warm water	Thermophilic actinomycetes
Japanese summer disease	Moldy houses	Various fungi
Machine worker's lung	Metal-cutting fluid	*Mycobacterium* species, Gram-negative bacilli
Malt worker's lung	Moldy malt	*Aspergillus* species
Mushroom worker's lung	Mushrooms	Mushroom spores, various other fungi
Peat moss worker's lung	Moldy peat moss	Various fungi
Sauna bather's lung	Sauna water	Various fungi
Sequoiosis	Moldy redwood dust	Various fungi
Suberosis	Cork	*Aspergillus* species, cork dust

Acute phase:

GGO – Consolidation – Centrilobular ground glass nodules

Mosaic attenuation + expiratory air trapping (headcheese sign)

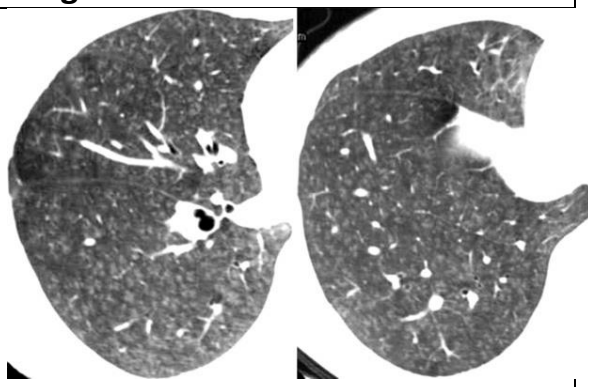
Chronic phase:

Interstitial reticular pattern => Pulmonary fibrosis (UL)

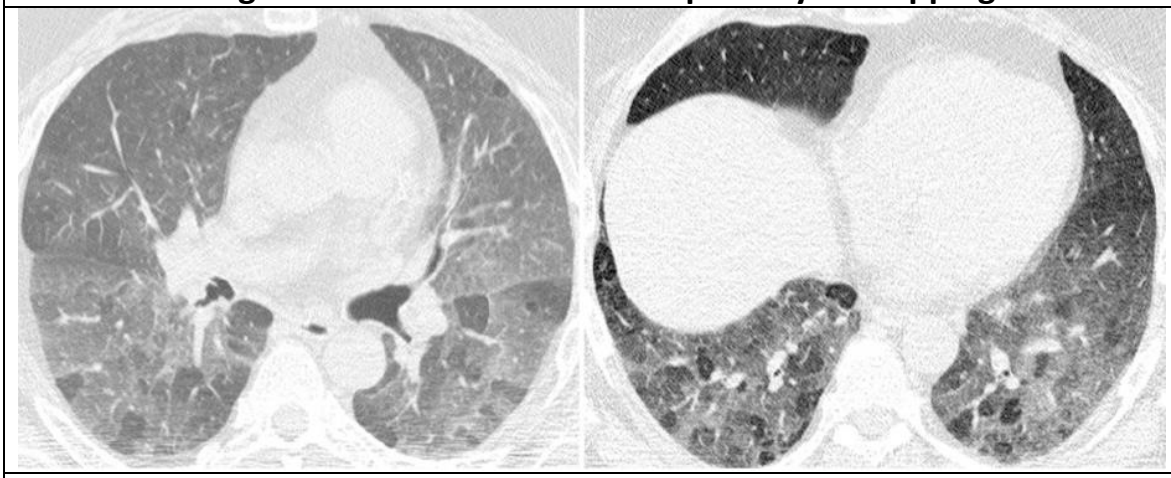
Honeycombing – Traction bronchiectasis – Architecture distortion

Distribution: patchy or diffuse central perihilar

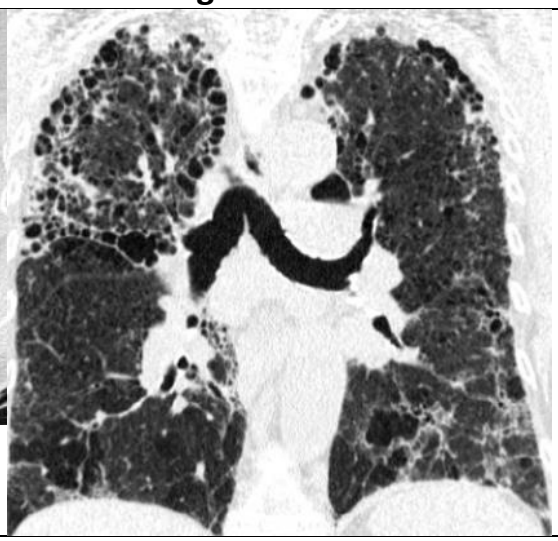
Centrilobular GGO + Centrilobular ground glass nodules



Headcheese sign = Mosaic attenuation + Expiratory Airtrapping



UL Fibrosis – Traction bronchiectasis - Honecombing



Interstitial Lung Diseases

Interstitial diseases due to:

- Thickening of interlobular septae
- Thickening of alveolar walls
- Thickening of pulmonary interstitium

Causes:

A) Edema: Interstitial Pulmonary edema
B) Infection: Interstitial Pneumonia
C) Granulomatous Disease: Sarcoidosis
D) Idiopathic: Idiopathic Interstitial Pneumonia
E) Collagen Vascular disease: RA – SLE – AS – Scleroderma
F) Pneumoconiosis: Asbestosis – Silicosis – CWP
G) Drugs
H) Radiation
I) Neoplastic: Lymphangitis Carcinomatosis

Approach:

1) Pattern: Reticular – Reticulonodular – GGO
2) Distribution: Upper vs Lower & Central vs Peripheral
3) Lung volume: Normal / Increased / Decreased
4) Evolution: Acute / Chronic
5) Pleural disease.
6) Mediastinal / hilar LNs

I- Idiopathic Interstitial Pneumonia (IIPs)

The American Thoracic Society–European Respiratory Society (ATS-ERS) classification of IIPs (2002) defines 7 morphologic patterns based on clinical-radiologic-pathologic diagnosis.

can be associated with collagen vascular disease, drug reaction, occupational exposure, or they may be idiopathic.

7 Morphological features:

1) **UIP = IPF = Usual interstitial pneumonia = Idiopathic pulmonary fibrosis**
2) **NSIP = Nonspecific interstitial pneumonia**
3) **COP = Cryptogenic organizing pneumonia**
4) **RB-ILD = Respiratory bronchiolitis–associated interstitial lung disease**
5) **DIP = Desquamative interstitial pneumonia**
6) **LIP = Lymphoid interstitial pneumonia**
7) **AIP = Acute interstitial pneumonia.**

ATS-ERS Criteria for Diagnosis of IPF in the Absence of Surgical Lung Biopsy

Major criteria

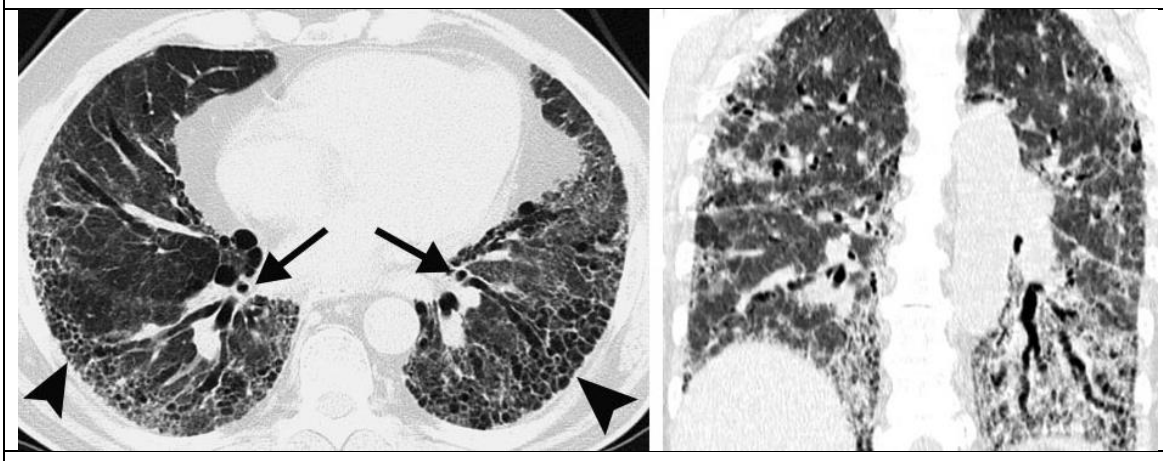
- Exclusion of other known causes of interstitial lung disease (eg, toxic effects of certain drugs, environmental exposures, and connective tissue diseases)
- Abnormal results of pulmonary function studies, including evidence of restriction and impaired gas exchange
- HRCT: Bibasilar reticular abnormalities with minimal GGO
- Transbronchial lung biopsy or BAL shows no features to support an alternative diagnosis

Minor criteria

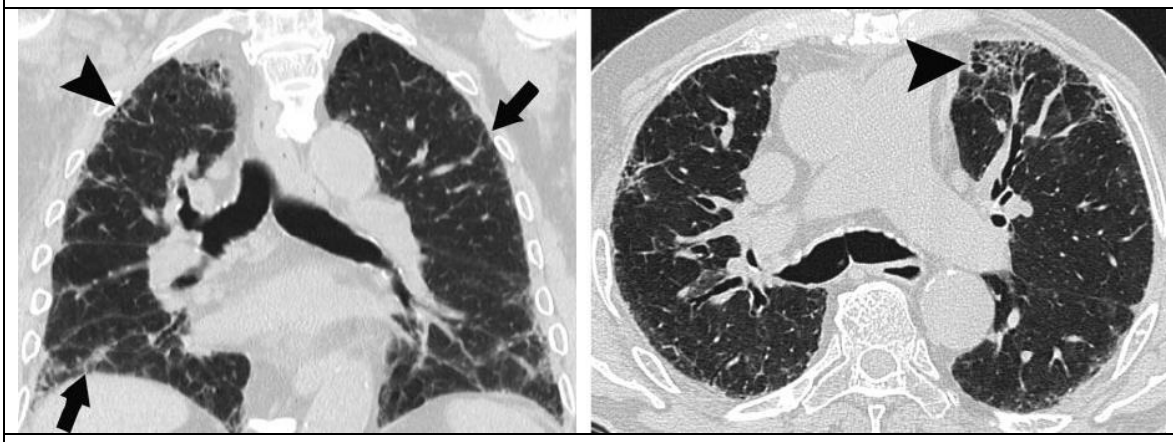
- Age > 50 y
- Insidious onset of otherwise unexplained dyspnea on exertion
- Duration of illness > 3 m
- Bibasilar inspiratory crackles (dry or “Velcro” type)

7 Morphological patterns	Associations	Distribution	HRCT pattern
UIP = IPF	**Idiopathic** **Collagen vascular diseases** **Drugs**	Peripheral & Basal	Reticular opacities Traction bronchiectasis Honeycombing
NSIP		Peripheral & Basal	GGO Reticular opacities
COP		Peripheral & Basal Migratory	Consolidation Reverse halo = Atoll sign: Central lucency is surrounded by a peripheral opacity.
RB-ILD	**Smoker**	UL	Centrilobular nodules Centrilobular emphysema
DIP		Peripheral & Basal	GGO
LIP	**AIDS** **Sjogren**	Basal	GGO Perivascular thin walled cysts
AIP = Diffuse alveolar damage	Idiopathic form of ARDS Preceded by viral infection /upper respiratory tract infection.	Basal	**<u>Exudative phase:</u>** GGO-Consolidation **<u>Organizing phase:</u>** Traction bronchiectasis Architecture distortion

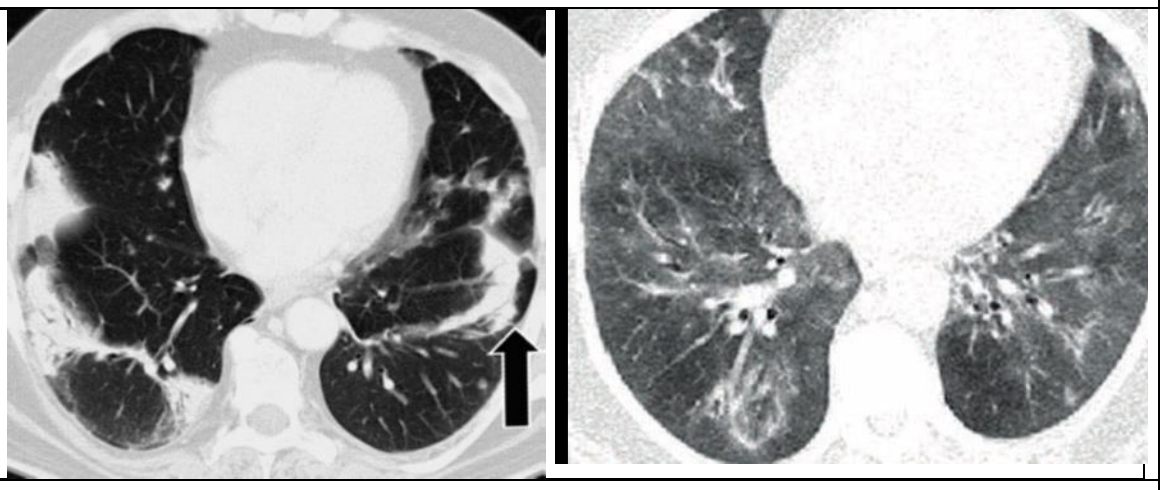
IPF

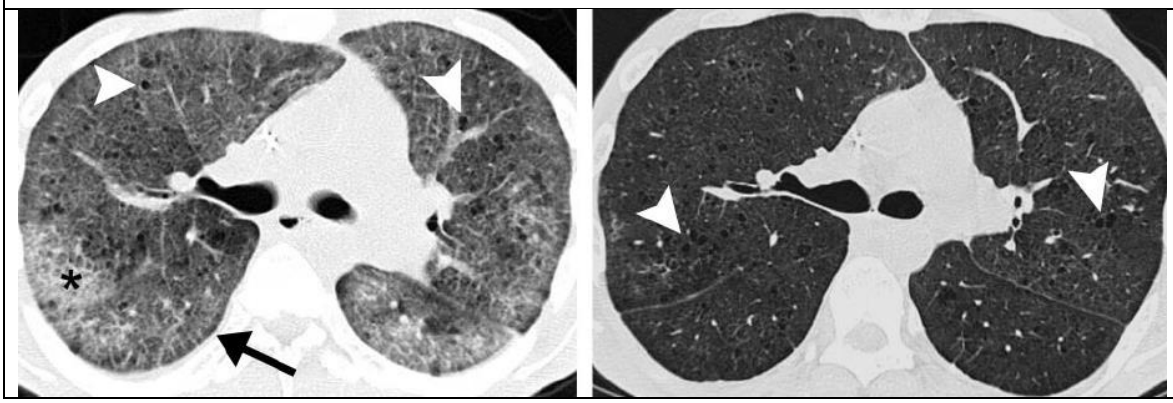


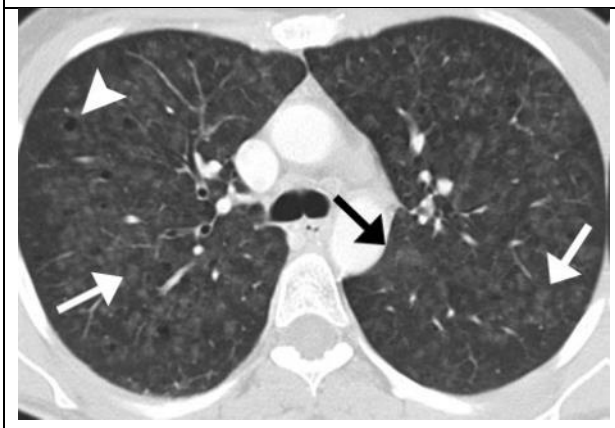
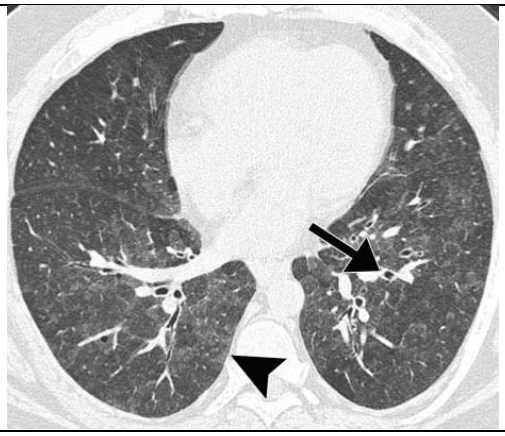
NSIP



COP



LIP


RB-ILD in Smoker	DIP in smoker
Bi UL	Bi basal
Centrilobular nodules	GGO
	

II- Collagen Vascular Disease

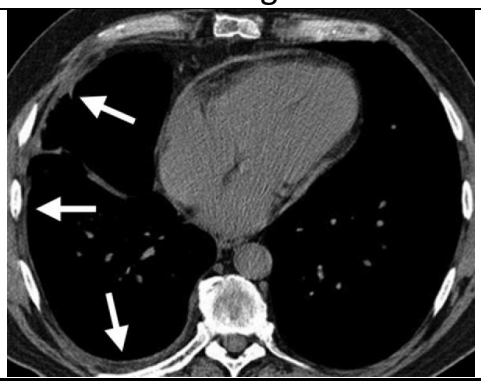
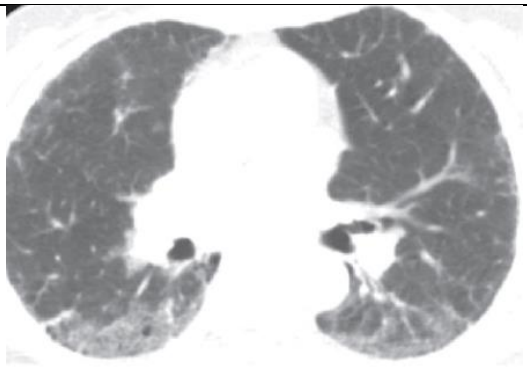
Immunologically mediated inflammatory systemic disorders that may affect various organs => Thoracic changes

Thoracic manifestations:

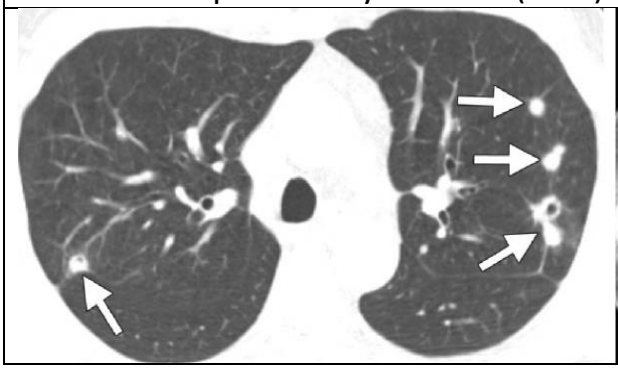
- **Interstitial lung disease (UIP – NSIP – COP – LIP)**
- **Pulmonary arterial hypertension (PAH)**
- **Pleural effusion / thickening**
- **Mediastinal lymphadenopathy**

1) Rheumatoid Arthritis

- F>m = 3:1
- Middle age
- Pleural: thickening and effusion (most common)
- NSIP = Bi basal GGO + septal thickening
- UIP= Bi basal & peripheral traction bronchiectasis + Honeycombing
- Pulmonary nodules + Cavitation

Pleural thickening & effusion	NSIP
	

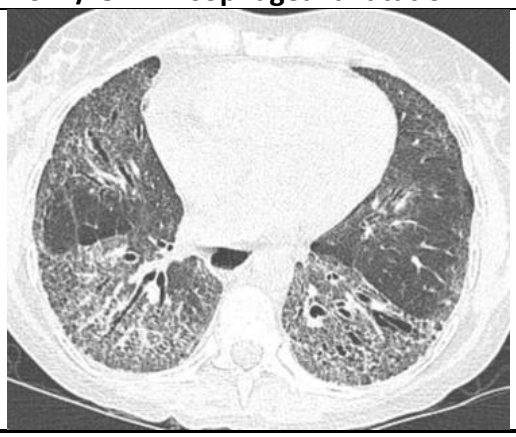
Caplan Syndrome = RA + Pneumoconiosis (Silicosis / CWP)
Rheumatoid pulmonary nodules (M>F): Cavitate + Calcify

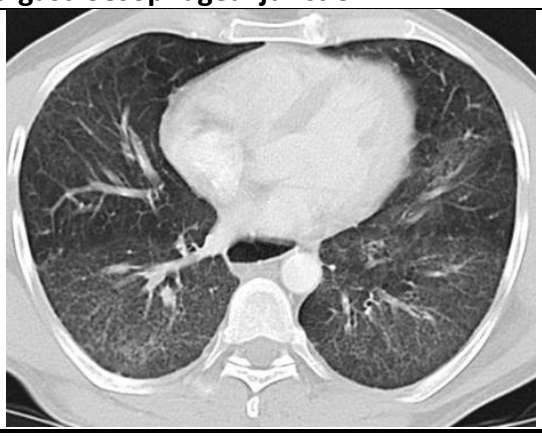


2) Progressive Systemic Sclerosis (PSS)

- F > M = 3:1
- NSIP = Bi basal GGO + septal thickening
- UIP = Bi basal & peripheral traction bronchiectasis + Honeycombing
- Esophageal abnormalities + GERD.

NSIP / UIP + Esophageal dilatation + Dilated gastroesophageal junction

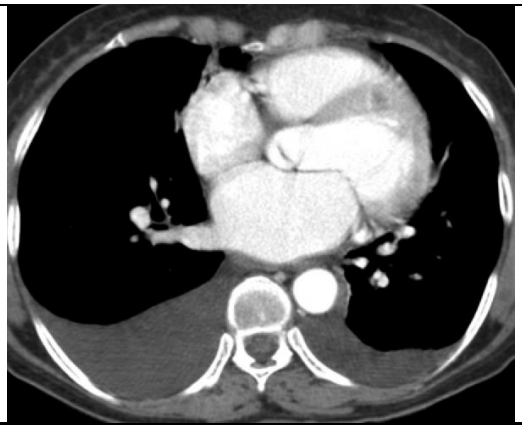




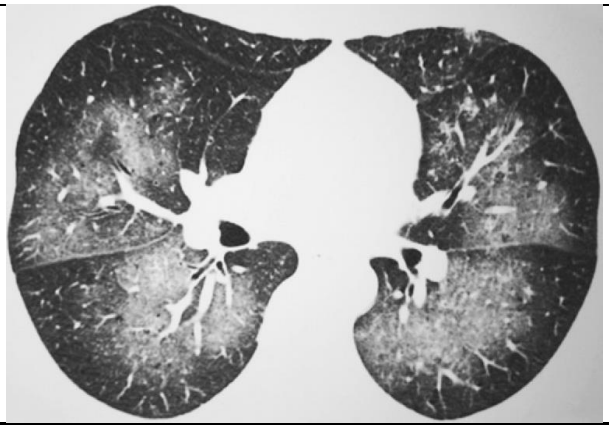
3) Systemic Lupus Erythematosus (SLE)

- Females
- Pleural disease: Thickening & effusion (Most common as RA)
- Pneumonia
- Acute lupus pneumonitis = Acute interstitial pneumonia (AIP): Bi LL Consolidation & GGO
- Diffuse alveolar hemorrhage (DAH): Bi central perihilar consolidation & GGO
- DVT + PE
- Shrinking lung syndrome: progressive loss of lung volume

Pleural effusion



DAH



4) Ankylosing Spondylitis (AS)

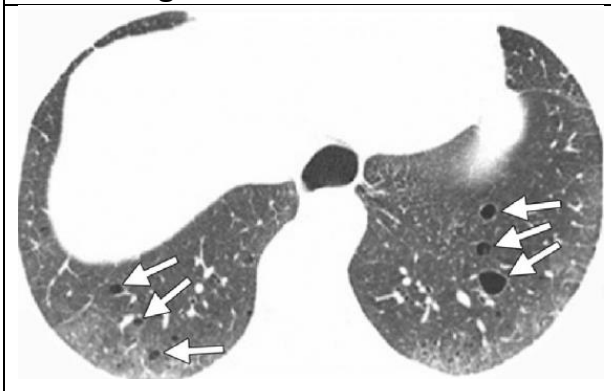
- M > F = 3:1
- Middle age males
- HLA-B27 antigen positive (90%)
- Bi & symmetrical sacroilitits + Spine disease

Upper lobe fibrosis
Cavitation
± Aspergilloma
(similar to TB)

5) Sjogren Syndrome

- Women + Dry eyes + Dry mouth
- LIP = GGO + Septal thickening + Perivascular cysts
- NSIP = Bi basal GGO + septal thickening
- UIP = Bi basal & peripheral traction bronchiectasis + Honeycombing
- Bronchiectasis + Mucoid impaction
- Pleural effusion: rare
- ↑ risk for Lymphoma

LIP = Thin walled cysts + GGO + Septal thickening



Bronchiectasis + Mucoid impaction

6) Polymyositis & Dermatomyositis

- Polymyositis = proximal muscle weakness
- Dermatomyositis = Skin manifestation + muscle weakness
- Respiratory failure: due to weakness of respiratory muscles or aspiration pneumonia
- COP = Bi patchy peripheral basal migratory Consolidation + GGO
- Atoll sign = crescent or ring shaped opacity + surrounded by GGO
- NSIP = Bi basal GGO + septal thickening
- UIP = Bi basal & peripheral traction bronchiectasis + Honeycombing
- ↑ risk of malignancy: Lung cancer most common

7) Mixed Connective Tissue Disease

manifestations of systemic lupus erythematosus, rheumatoid arthritis, progressive systemic sclerosis, and polymyositis or dermatomyositis are combined.

The disease is characterized by the following clinical and laboratory findings: Raynaud phenomenon, swollen hands, arthritis or arthralgia, acrosclerosis, esophageal dysmotility, myositis, pulmonary hypertension, a high titer of speckled fluorescent antinuclear antibodies, and the presence of a moderate to high serum level of antibodies against ribonucleoprotein

Middle age females

- NSIP = Bi basal GGO + septal thickening
- UIP = Bi basal & peripheral traction bronchiectasis + Honeycombing
- Pleural thickening or effusion

III- Pneumoconiosis

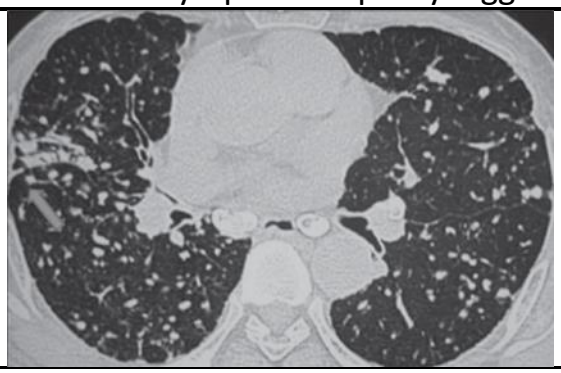
Pneumoconiosis is a lung disease secondary to inorganic dust inhalation.
Hypersensitivity pneumonitis is caused by organic dust inhalation.

1) Silicosis

due to inhalation of silica dust, which miners may be exposed to.

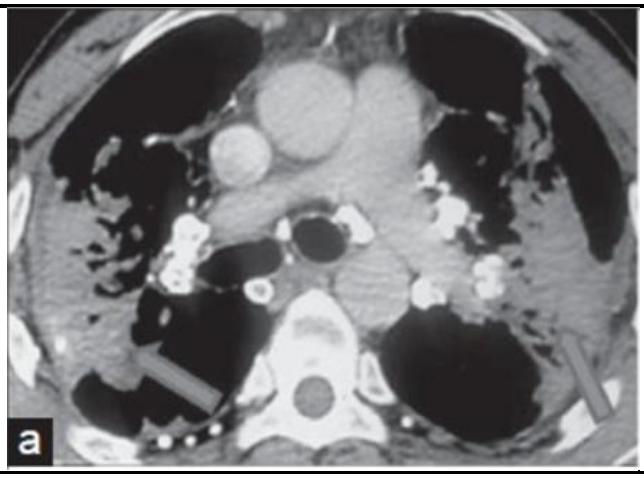
Chronic Silicosis:

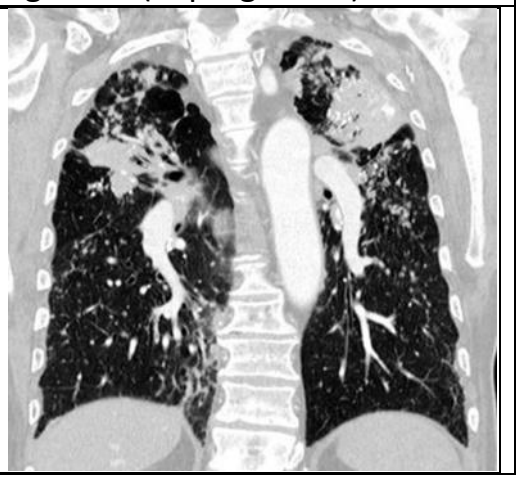
- 20-40y of exposure
- Pattern:
  - Nodular pattern: Calcification 20%
  - Reticulo-nodular pattern
- Distribution: UL > LL
- Hilar lymphadenopathy: Egg shell calcification (5%)



Progressive Massive Fibrosis (PMF):

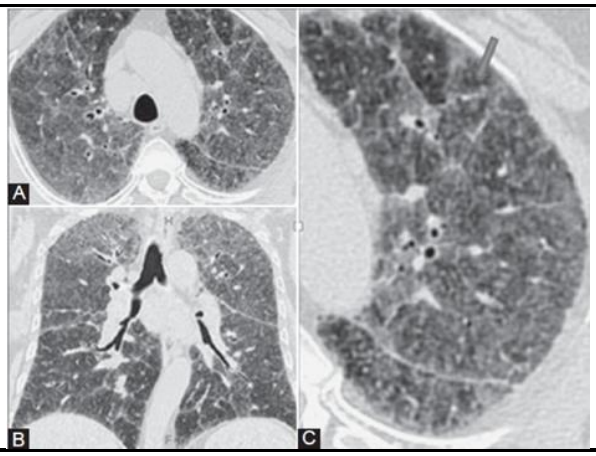
- UL posterior segment
- Conglomerate masses formed by coalescent nodules
- Vertical orientation
- Fibrosis + Cavitation => TB / Fungal ball (Aspergilloma)





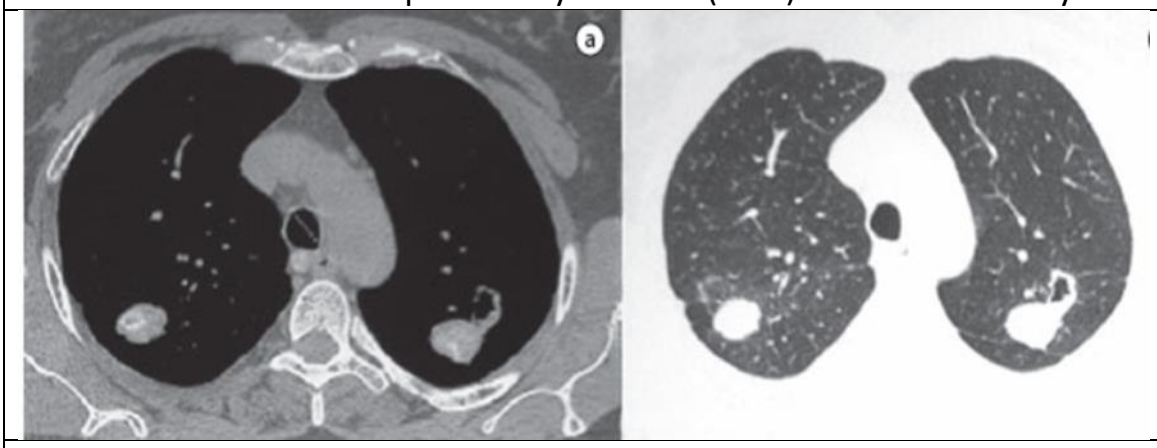
Acute Silicosis:

- <3y of exposure
- Fulminant course
- Diffuse / multifocal
- Air space opacities: Consolidation – GGO + Septal thickening



Caplan Syndrome:

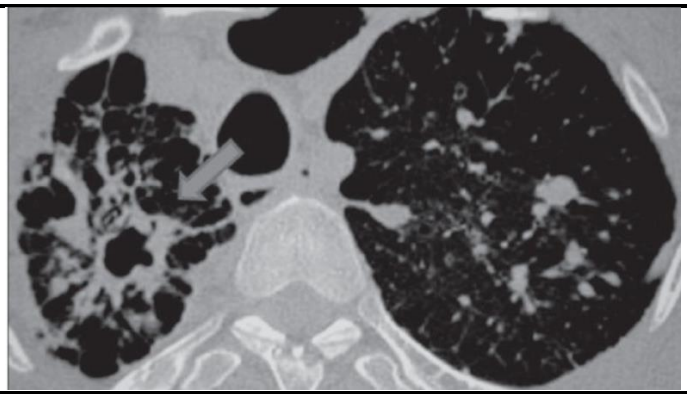
- RA + Silicosis (less common) / CWP (more common)
- Rheumatoid pulmonary nodules (M>F): Cavitate + Calcify



Silicotuberculosis:

- Silicosis + TB (25%)

- TB= UL cavity + Fibrosis
- Silicosis = Multiple nodules



2) Coal Workers Pneumoconiosis (CWP)

caused by inhalation of coal dust, which does not contain any silica

Radiologically indistinguishable from silicosis

3) Asbestosis:

caused by inhalation of asbestos fibers

A) Asbestosis – Related Pleural Disease (5):

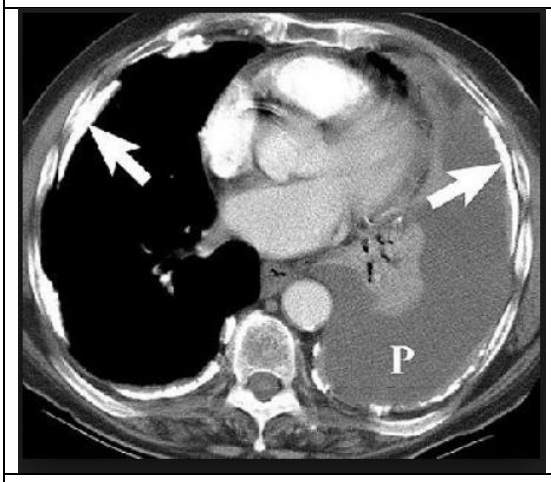
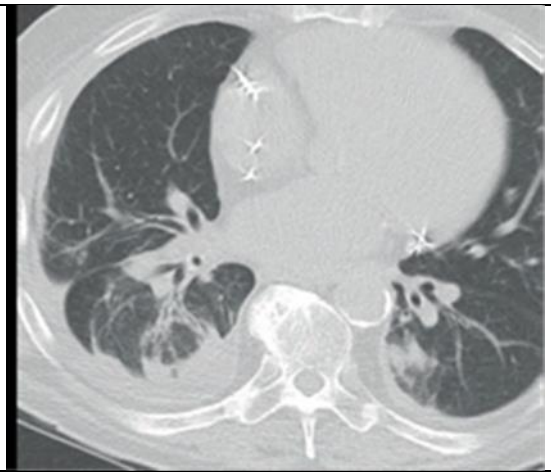
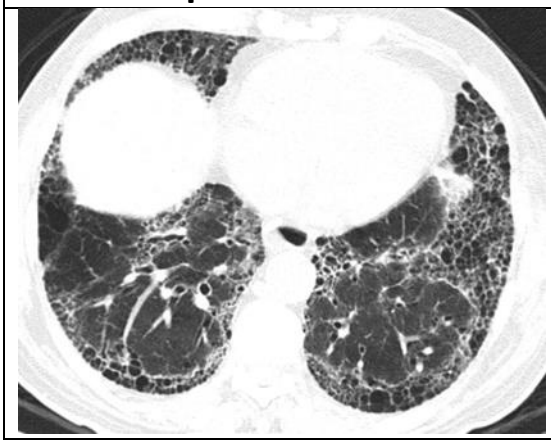
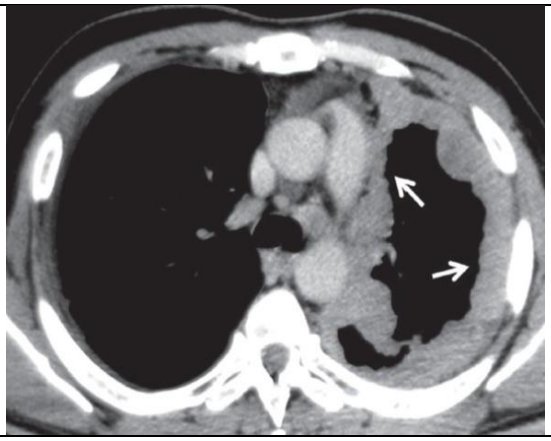
1) Pleural plaques (most common)
    - Central – Posterolateral
    - Mid – Lower chest
    - Focal + Interrupted + pleural thickening + Sharp margins
    - ± Calcification
2) Diffuse pleural thickening
3) Pleural calcifications
    - DD: TB – Empyema – Hemothorax – Surgery
4) Benign pleural effusion
5) Round atelectasis:
    - Abnormal pleural: pleural thickening – effusion – plaques
    - Peripheral pleural based opacity
    - Rounded / elliptical opacity
    - Volume loss in the affected lobe.
    - Comet tail sign = curved pulmonary vessels and bronchi from hilum leading into the opacity

B) Asbestosis – Related Interstitial Pulmonary Fibrosis:

- Pattern: Reticular pattern (Similar to UIP = IPF):
    - Smooth septal thickening - Parenchymal bands
    - Traction bronchiectasis - Honeycombing
- Distribution: Bi – Basal – Peripheral / Subpleural
- No significant lymphadenopathy

C) Asbestosis – Related Malignancy:

- Increased risk of:
    - Mesothelioma (latency >30y after exposure)
    - Bronchogenic carcinoma
    - GIT neoplasms

Pleural Calcifications	Round Atelectasis
	
Reticular pattern	**Mesothelioma**
	

IV- Pulmonary Sarcoidosis

Idiopathic systemic chronic granulomatous non-caseating disease of unknown etiology

Thoracic involvement in 90% of patients

F > M
Middle age
Black > White

Diagnosis

Clinical + Radiological + Histological
Biopsy: Transbronchial biopsy, CT-guided biopsy, or surgical biopsy
Kveirn test

Stages (Silzbach Classification) & Typical HRCT features

- Stage 0: Normal (5%)
- Stage 1: Lymphadenopathy (50%): (Garland's triad)
    - Bilateral & symmetrical hilar lymphadenopathy
    - Mediastinal: Right paratracheal + Tracheobronchial + Azygos
    - Egg shell calcification (5%)
- Stage 2: Lymphadenopathy + Pulmonary infiltration (30%):

    Pattern: Reticulo-nodular infiltration
    - Nodules:
        - Micronodules (2–4 mm; well defined, bilateral)
        - Macronodules (≥5 mm, coalescing)
        - Lymphatic distribution: peribronchovascular, subpleural, interlobular septal
    - Fibrotic changes: reticular opacities, architectural distortion, traction bronchiectasis, bronchiolectasis, volume loss

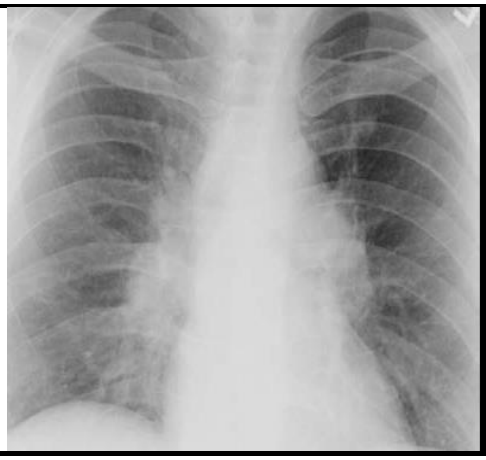
    Distribution: Predominant upper and middle lung zones
- Stage 3: Pulmonary infiltration + No lymphadenopathy (10%)
- Stage 4: Upper lobe pulmonary fibrosis ± Bullae - Fungal ball (5%)

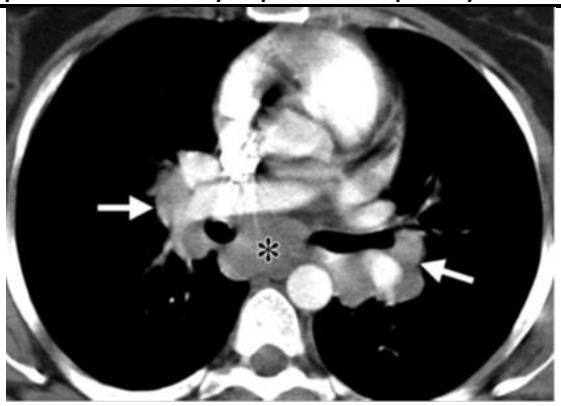
Atypical features

- Lymphadenopathy: unilateral, isolated, anterior and posterior mediastinal
- Alveolar sarcoid pattern: Airspace consolidation – GGO
- Linear opacities: interlobular septal thickening, intralobular linear opacities

- Fibrocystic changes: cysts, bullae, blebs, emphysema, honeycomb-like opacities with upper- and middle-zone predominance
- Miliary opacities
- Airway involvement: mosaic attenuation pattern, tracheobronchial abnormalities, atelectasis
- Pleural disease: effusion, chylothorax, hemothorax, pneumothorax, pleural thickening, calcification, plaques
- Mycetoma, aspergilloma

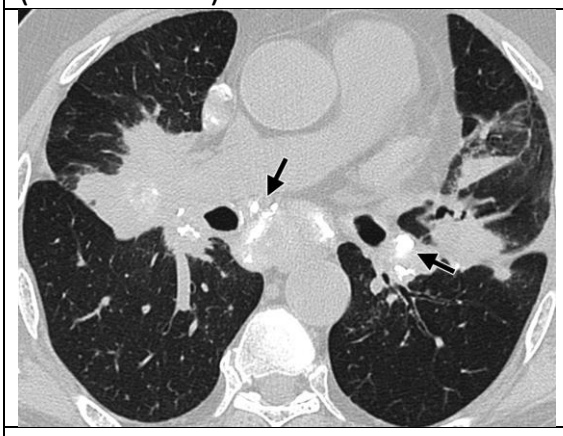
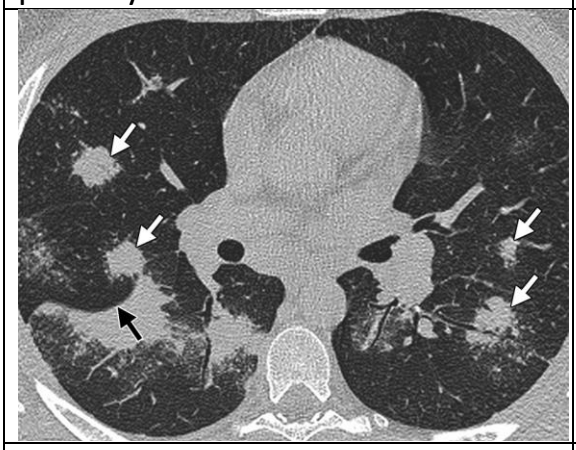
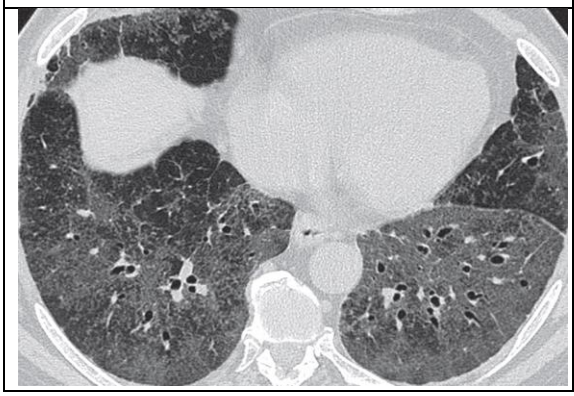
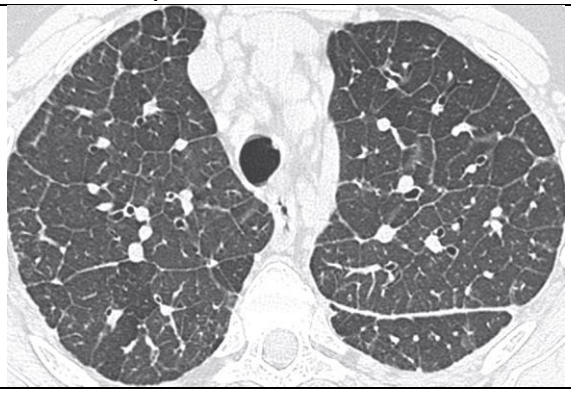
Garland's triad = Bilateral hilar + Right paratracheal lymphadenopathy





Reticulo-nodular pulmonary infiltration

UL pulmonary fibrosis + traction bronchiectasis + Bullae

Conglomerate masses and eggshell-like nodal calcifications (DD: Silicosis)	
	
Sarcoid galaxy sign = Fine nodular opacities around the large nodules (white arrows), Small low-attenuation spots that correspond to the spaces between partially coalescent small nodules	Alveolar sarcoid pattern of airspace consolidation
	
GGO	Reticular pattern
	

V- Lymphangitis Carcinomatosis

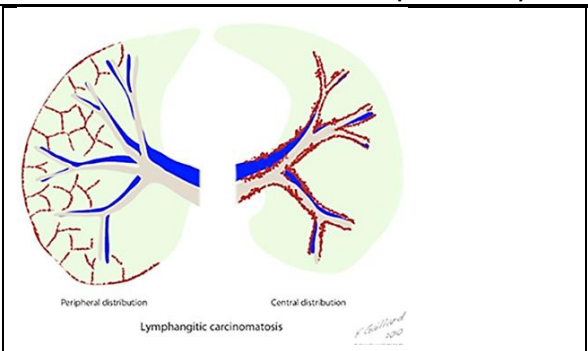
Tumor spread through the lymphatics of the lung

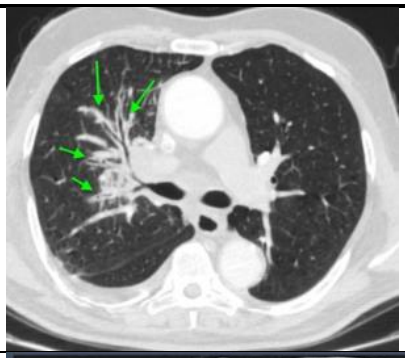
Secondary to adenocarcinomas such as:

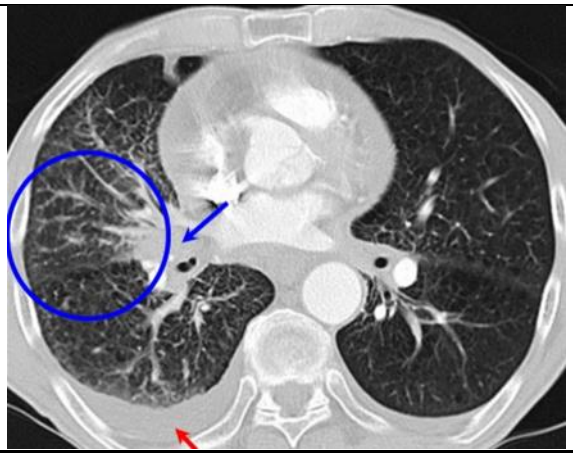
- Breast cancer - most common
- Lung cancer (Bronchogenic adenocarcinoma)
- Colon cancer
- Stomach cancer
- Prostate cancer
- Cervical cancer
- Thyroid cancer

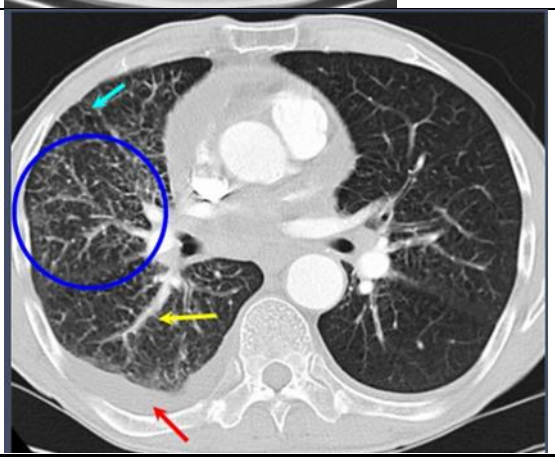
Radiological features:

→ Interlobular septal thickening: Nodular > Smooth
→ Peribronchial thickening: Nodular / Irregular
→ Fissural thickening
→ Subpleural nodules
→ Hilar and mediastinal nodal enlargement (40-50%)
→ Pleural effusion (30-50%)









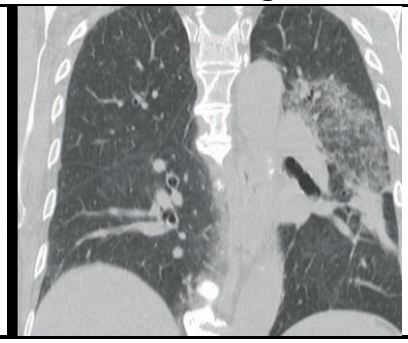
Iatrogenic Lung Diseases

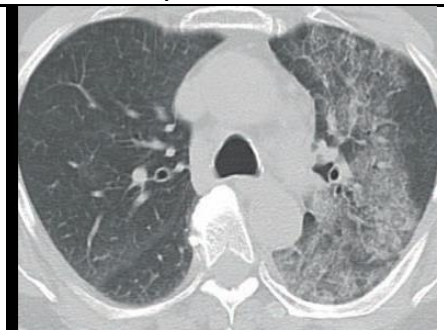
Drug Toxicity

- Pulmonary drug reaction, most commonly to cytotoxic drugs.
- The lung has a diverse responses to injury:
  - ⇨ Pulmonary edema (due to increased capillary permeability)
  - ⇨ ARDS
  - ⇨ Organizing pneumonia
  - ⇨ Eosinophilic pneumonia
  - ⇨ Bronchiolitis obliterans
  - ⇨ Pulmonary hemorrhage
  - ⇨ NSIP and UIP.

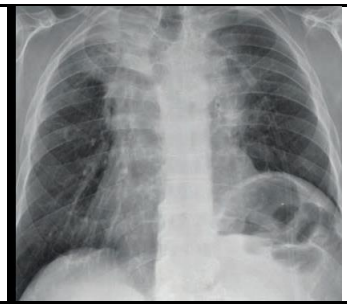
Radiation Lung Injury

- The radiographic abnormality is largely confined to the radiation port, usually with non-anatomic linear margins.
- Early stage (1-3m after radiotherapy) => Radiation pneumonitis
  - Ground glass centered on the radiation port.





- Late stage (6-12m after radiotherapy) => Radiation fibrosis
  - Pulmonary fibrosis
  - Traction bronchiectasis
  - Architecture distortion



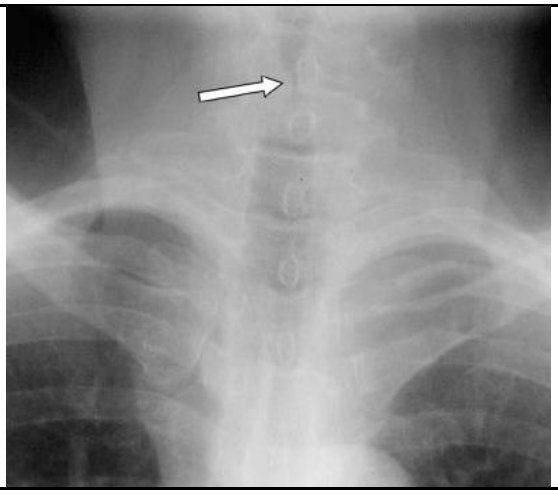
Large Airway Diseases

I- Non-Neoplastic Tracheal Stenosis/Wall Thickening

A) Focal Tracheal Stenosis:

1) Post-intubation:

Subglottic region
Symmetric narrowing
< 2 cm in length
Hourglass shape

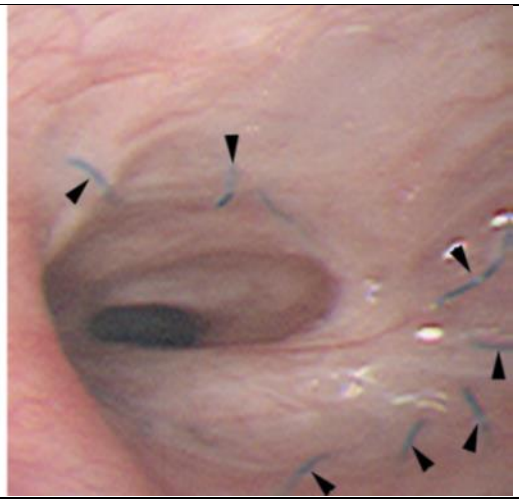


2) Post-infection: TB

Focal narrowing of the trachea or bronchi
Calcification of the stenotic segment is rare.

3) Post- Transplantation Stenosis:

Fibrotic, focal, cicatricial narrowing at the bronchial anastomotic site.



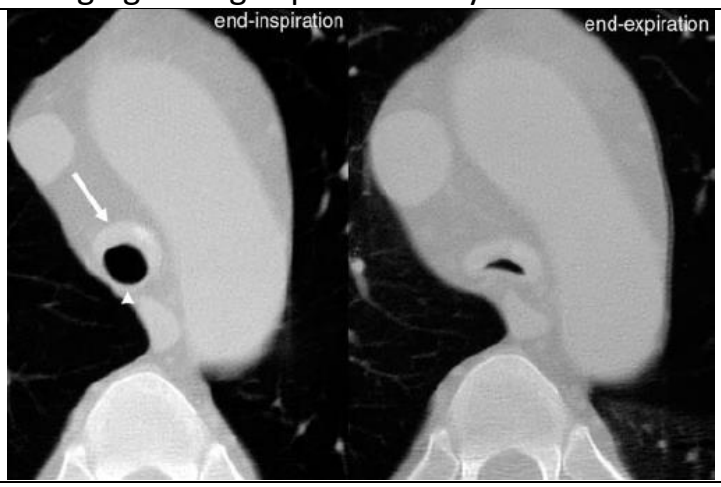
4) Post-inflammation: Crohn's disease

B) Diffuse / Multifocal Tracheal Stenosis:

i) Sparing posterior wall:

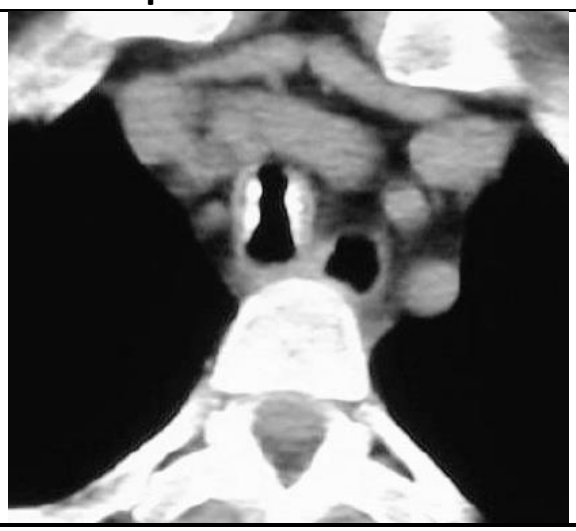
1) Relapsing Polychondritis:

- Middle-aged women.
- Multisystemic autoimmune disease of unknown etiology
- Recurrent inflammation + Progrossive destruction of cartilaginous structures:
  - Nose, ear, joints, larynx, trachea, and bronchi
- Recurrent chondritis of auricle (painfule ear)
- Cochlear & vestibular damage
- Smooth thickening of the cartilaginous trachea/bronchial walls
- No calcification
- Dynamic imaging during expiration may demonstrate airway collapse



2) Tracheobronchopathia Osteochondroplastica:

- Idiopathic benign condition
- Lower 2/3 trachea + proximal portions of primary bronchi
- Calcified/ossified submucosal nodules along the tracheal walls



ii) Circumferential wall thickening:

1) Wegener granulomatosis:

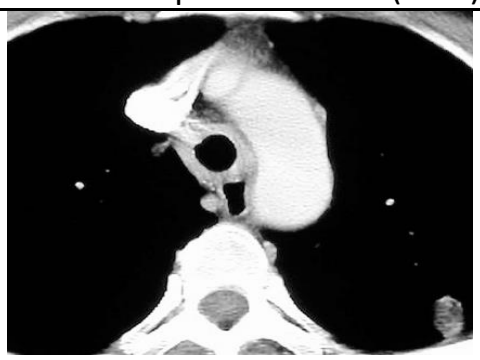
- Systemic necrotizing non-caseating granulomatous small-vessel vasculitis
- Classic clinical triad = Sinusitis + Lung involvement + Renal insufficiency
- C-ANCA is positive.

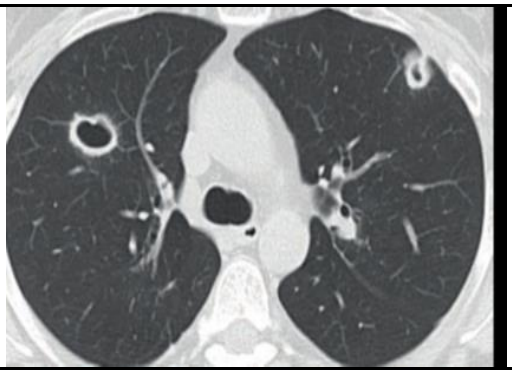
Upper Respiratory Tract:

- Nasal ulceration
- Sinusitis: granulomatous masses + bone destruction
- Tracheal circumferential nodular wall thickening + No calcification + Posterior wall involvement

Pulmonary Involvement:

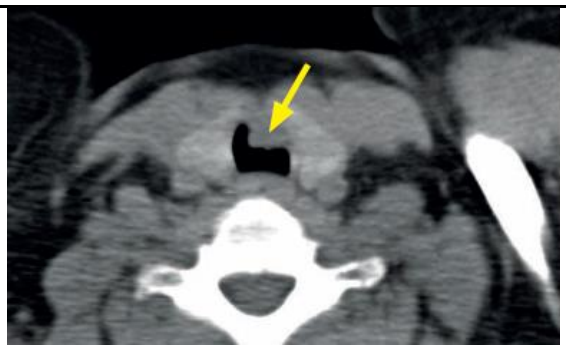
- Peripheral subpleural cavitary pulmonary nodules
- Bilateral perihilar GGO (DAH)

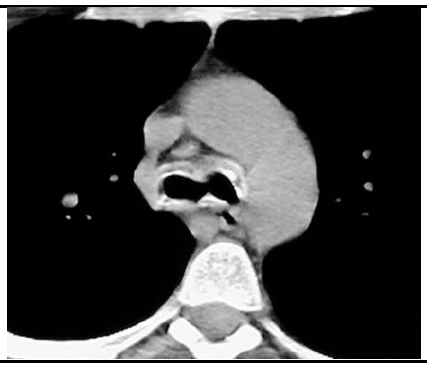




2) Amyloidosis:

- Characterized by deposition of abnormal proteinaceous material (amyloid) in extracellular tissue.
- It can be idiopathic or associated with various inflammatory, hereditary, or neoplastic pathogeneses.
- Nodular and irregular tracheal wall thickening
- Calcified/ossified nodules
- Posterior wall involvement (DD:Tracheobronchopathia Osteochondroplastica)



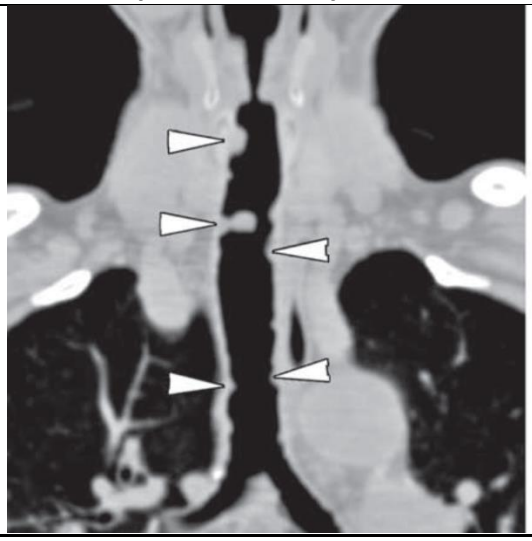


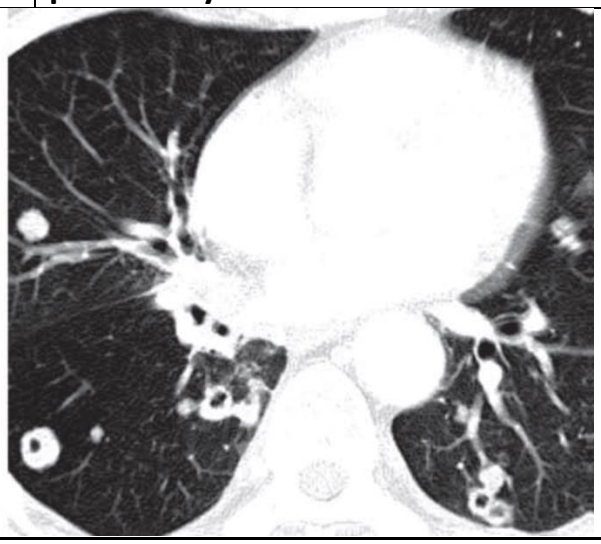
3) Papillomatosis:

- Infection of the upper respiratory tract by the human papillomavirus.
- Infection occurs most frequently at the time of birth, when the child passes through an infected birth canal.
- Multiple Laryngeal/Tracheal/Bronchial polypoidal nodules
- Multiple pulmonary cavitating nodules.

Multiple polypoid nodules in trachea (arrowheads)

Multiple well-defined cavitating pulmonary nodules



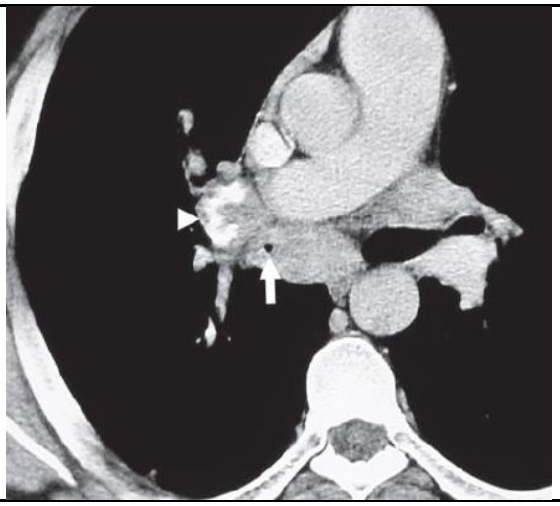


4) Rhinoscleroma

- Chronic, progressive, granulomatous infection that affects the respiratory tract from the nose to the bronchi.
- The etiologic agent is a gram-negative: Klebsiella rhinoscleromatis

5) Sarcoidosis:

- Narrowing of the airway may be secondary to:
  - Extrinsic compression from enlarged lymph nodes
  - Granuloma formation within the airway mucosa and submucosa.



II- Tracheal Widening / Tracheomalacia

Increase in tracheal diameter +
Increase in compliance (Tendency to collapse on expiration)

Acquired causes:

- **Ageing**
- **COPD/Chronic bronchitis / Emphysema / Asthma**
- **Prolonged intubation, especially in those with tracheostomy**
- **Pulmonary fibrosis**

Congenital causes:

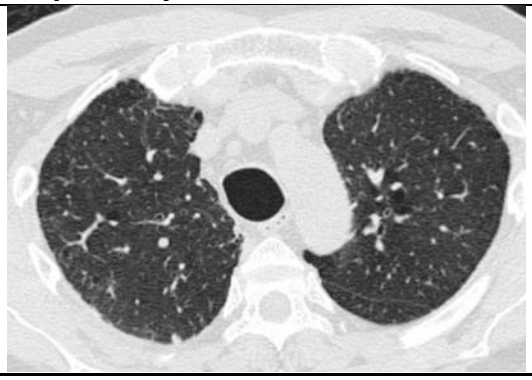
- Cystic fibrosis
- **Mounier-Kuhn syndrome (tracheobronchomegaly)**
    - Idiopathic
    - Associated with:
        - Ehlers-Danlos syndrome (in adults)
        - Cutis laxa (in children)
- Marfan syndrome
- Kenny-Caffey syndrome
- Cornelia-de Lange syndrome
- Bruton-type agammaglobulinemia

Inspiratory CT:

⇨ Dilated trachea (> 3 cm)

Expiratory CT:

⇨ Anterior bowing of the posterior membranous portion
⇨ Crescent shape in the axial plane
⇨ Decrease of anterior-posterior diameter > 50%

Inspiratory CT	Expiratory CT
	

III- Airway Tumors

Primary tumors of the trachea and main bronchi are **rare**
1% to 2% of all respiratory tract tumors.

- **In adults => majority are malignant tumors**
- **In children => majority are benign tumors**

Classification of Tracheobronchial Tumors

Primary malignant tumors
- Surface epithelium
  - SCC (most common), adenocarcinoma, neuroendocrine tumors (carcinoid tumor, large cell neuroendocrine tumors, small cell carcinoma)
- Salivary glands
  - ACC (second most common), MEC
- Mesenchyme
  - Sarcoma, malignant lymphoma

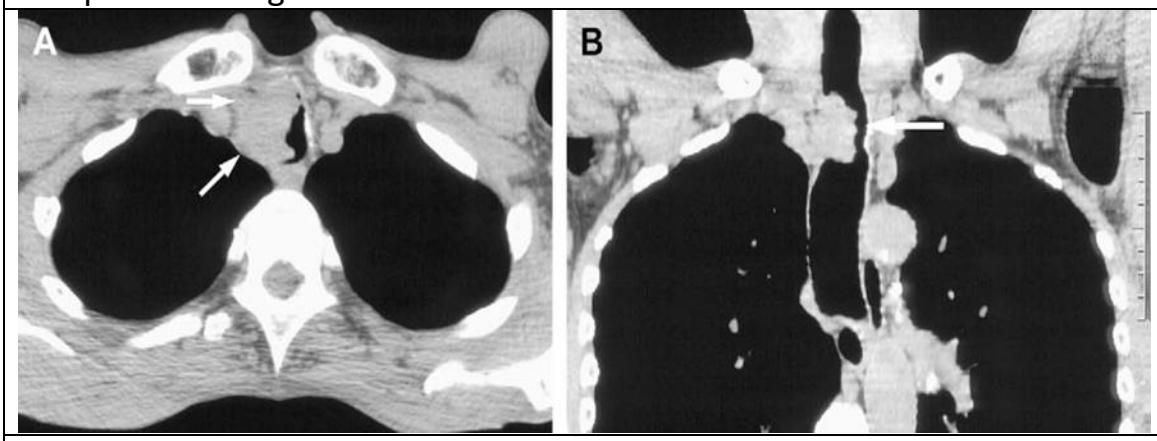
Benign tumors
- Surface epithelium
  - Squamous cell papilloma, papillomatosis, salivary glands, pleomorphic adenoma, mucous gland adenoma, oncocytoma
- Mesenchyme
  - Hamartoma, leiomyoma, lipoma, fibroma, neurogenic tumor

Secondary malignant tumors
- Direct invasion (most common)
  - Thyroid cancer, laryngeal cancer, lung cancer, esophageal cancer
- Hematogenous metastasis
  - Melanoma, breast cancer, renal cell carcinoma, colon cancer

❖ Malignant Tumors

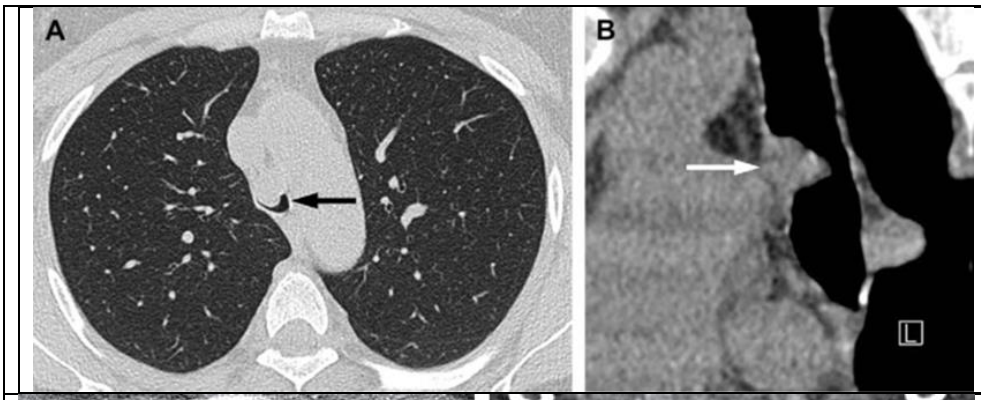
1) Squamous Cell Carcinoma (SCC):

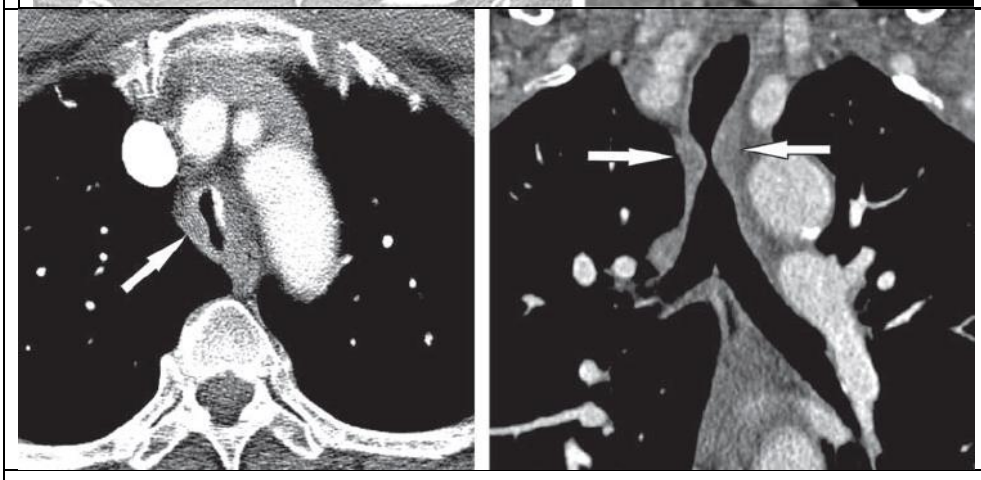
- is most frequent primary malignancy of the trachea,
- M> F = 4:1
- Age = 50 - 60y.
- Strongly associated with habitual cigarette smoking.
- A large polypoid intraluminal soft tissue mass within the central airways with either exophytic or ulcerative component.
- Eccentric narrowing of the airway lumen
- Regional extent into the esophagus => Tracheoesophageal fistula
- Spreads to regional LNs



2) Adenoid Cystic Carcinoma (ACC)

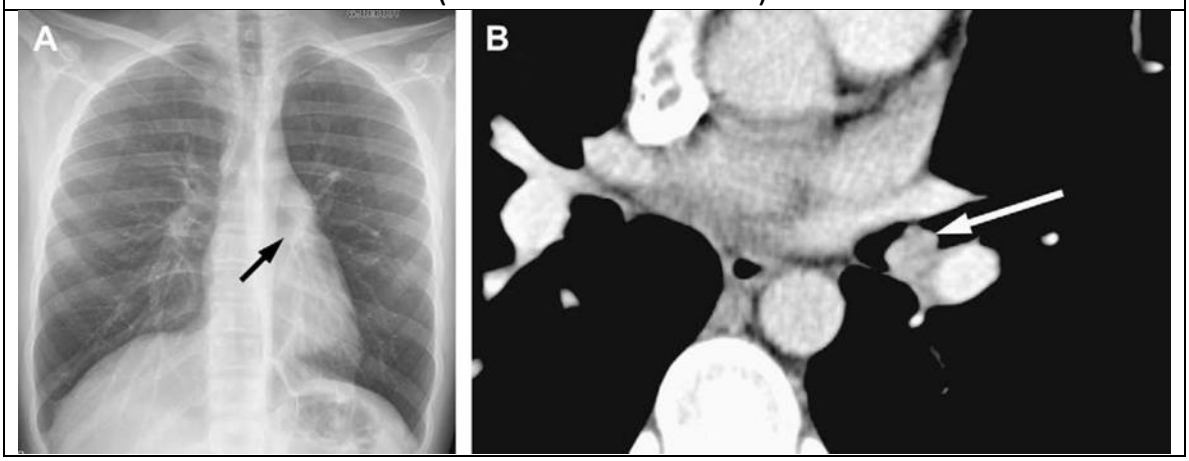
- 2nd most common tracheo-bronchial tumors
- Low-grade malignancy formerly named cylindroma
- not associated with cigarette smoking.
- M = F
- AGE = 40y
- arises from the salivary glands lining the mucosa of the airways
- Most of the tumors arise in the lower trachea or main stem bronchi
- A well-defined soft-tissue intraluminal mass
- Grows slowly infiltrate the wall of the airways
- Spread along submucosal and perineural planes.
- Rarely associated with regional lymph node metastases.





3) Mucoiepidermoid Carcinoma

- Rare tumor that originates from minor salivary glands lining the tracheobronchial tree.
- Age: < 40y
- CT: Endobronchial mass (DD: Carcinoid tumor)



❖ Neuroendocrine Tumors

4 Types:

a. Carcinoid Type1 (Typical)
b. Carcinoid Type 2 (Atypical)
c. Large cell neuroendocrine tumors (LCNE)
d. Small cell carcinoma (SCC)

WHO Classification:

A. Well-differentiated tumors = Typical Carcinoid tumors = Low-grade
B. Well-differentiated tumors = Atypical carcinoid tumors = Intermediate grade = more aggressive + metastases.
C. Poorly differentiated tumors = Small cell carcinoma + Large cell carcinoma = high-grade malignancies with a poor prognosis

Location:

GIT carcinoid (85%)

- Appendiceal carcinoid - 50% of all carcinoid tumours
- Small bowel carcinoid
  - Terminal ileum ~ 90% of all small bowel carcinoid tumours
- Gastric carcinoid

Bronchopulmonary carcinoid (20-30%):

- Central bronchial carcinoid (Main, lobar or segmental bronchi) (60%)
- Peripheral pulmonary carcinoid tumors (40%)
- Tracheal location: very rare

Primary hepatic carcinoid
Ovarian carcinoid (0.5% of carcinoid tumours)
Thymic carcinoid

Bronchopulmonary Carcinoid Tumors

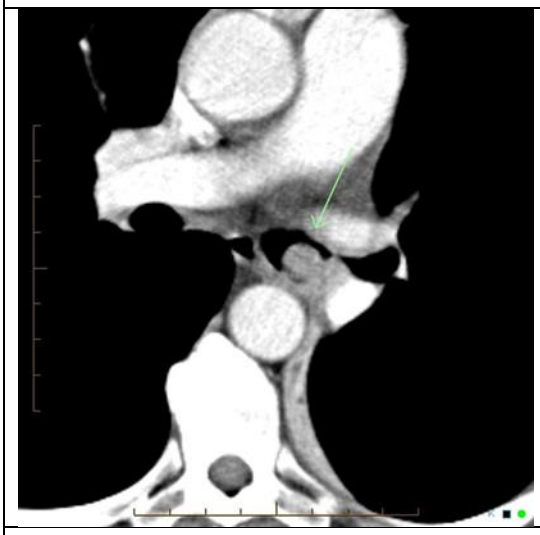
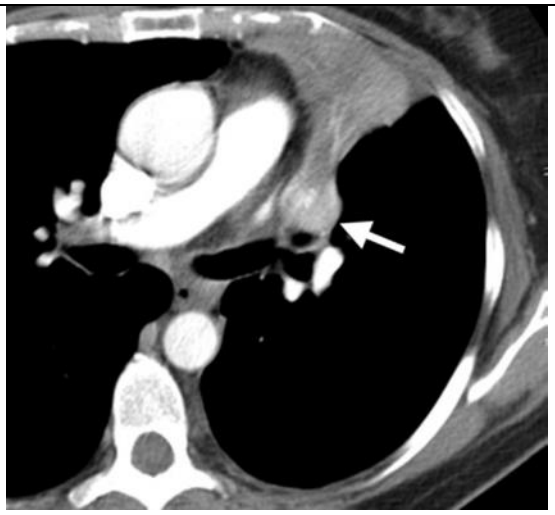
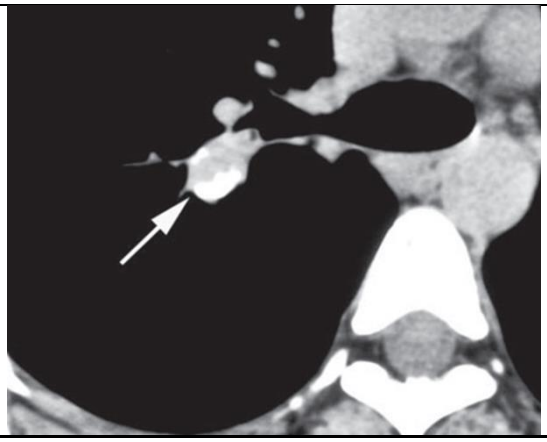
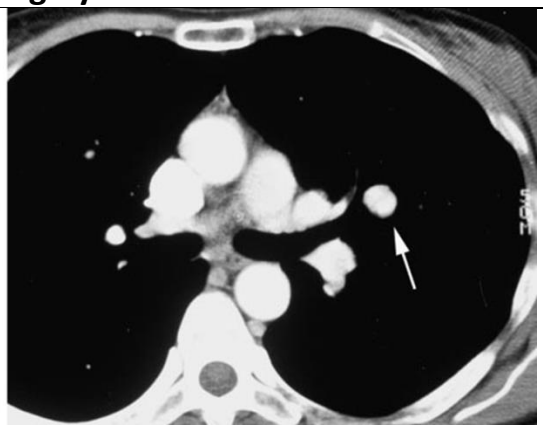
- Most frequent primary pulmonary neoplasm in childhood
- Age: Middle age
- Sex: M = F
- Typical (Type I) > Atypical (Type 2)
- Central location > Peripheral location

Location:

- Central: bronchial carcinoid (Main, lobar or segmental bronchi) (60%)
- Peripheral: pulmonary carcinoid tumors (40%)
- Tracheal location: very rare

Radiological Features:

- A hilar or perihilar mass which is usually well-defined, round or ovoid.
- Small tumors may be entirely intrabronchial
- Marked homogeneous contrast enhancement due to high vascularity.
- Calcifcation (usually eccentric)
- Distal atelectasis / pneumonia

Endobronchial lesions	**Distal atelectasis**
	
Eccenteric Calcification	**Highly vascular**
	

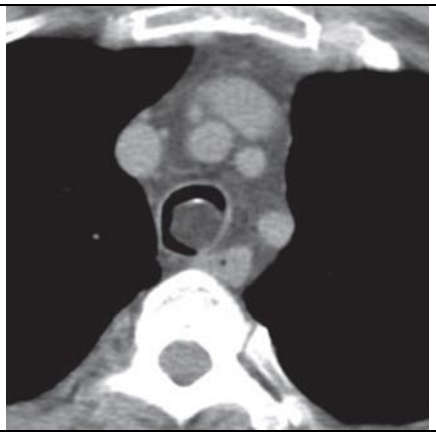
❖ Benign Tumors:

1) Endobronchial Hamartoma

Well defined endobronchial lesion.
Fat + calcifications within the lesion

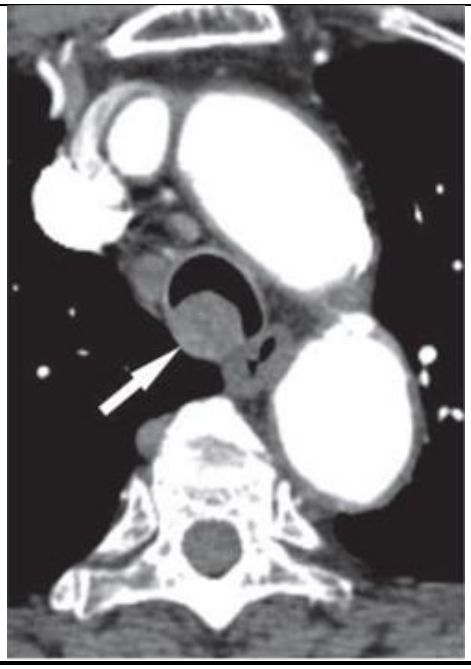
2) Endobronchial Lipoma:

Well defined endobronchial lesion.
Fat attenuation within the lesion

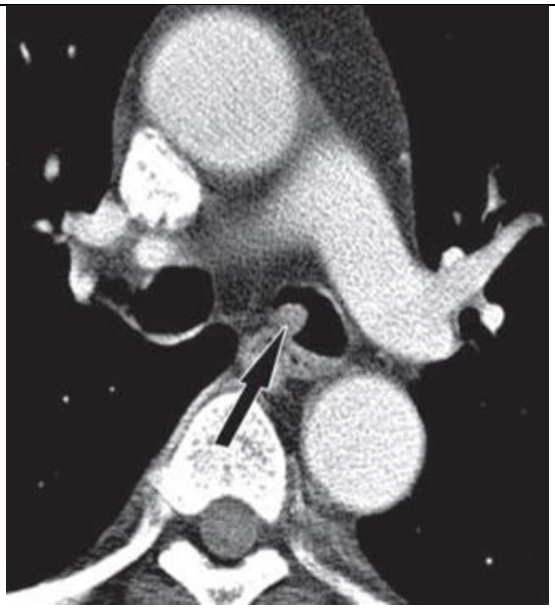


3) Endotracheal Leiomyoma:

- Rare benign neoplasm of the trachea-bronchial tree
- Well defined endobronchial lesion
- Homogenous density and enhancement
- No wall destruction



4) Endobronchial Schwannoma



5) Mucoid Impaction

- Bronchus filled with inspissated mucus (Soft tissue density)
- Bronchi may be enlarged (Bronchocele) or of normal size.
- Finger in glove sign = tubular or branching opacities; radiate from the hilum toward the periphery of the lung
- No contrast enhancement

Causes of mucoid impaction:

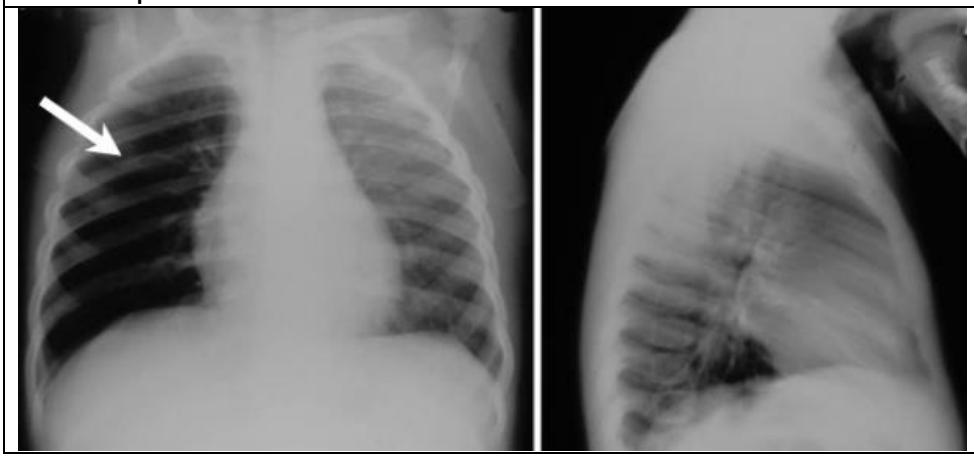
Congenital Segmental bronchial atresia Cystic fibrosis Inflammatory-infectious ABPA in asthma or cystic fibrosis* Broncholithiasis Foreign body aspiration	Neoplastic Benign Bronchial hamartoma Lipoma Papillomatosis Malignant Bronchogenic carcinoma Carcinoid tumor Metastases

6) FB

- Children.
- 80% of aspirated foreign bodies will be non-opaque

<u>Air trapping leading</u>

- Obstructive emphysema
- Lobar / segmental Atelectasis
- Post-obstructive pneumonia
- Abscess
- Mediastinal shift
- Complication: Pneumomediastinum



❖Secondary Tracheal Malignancy

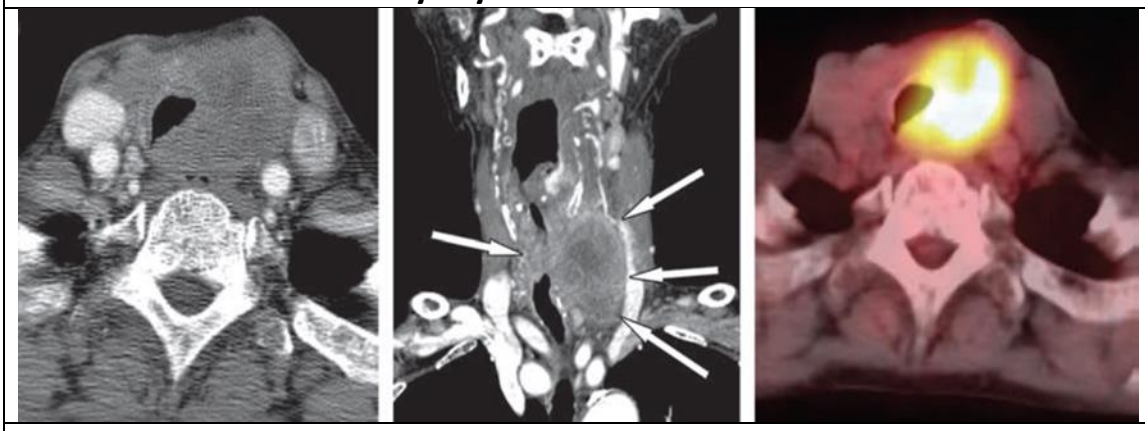
A) Direct Invasion

Direct invasion of the central airways by neoplasms of the thyroid, esophagus, lung, and larynx is much more frequent than hematogenous metastases

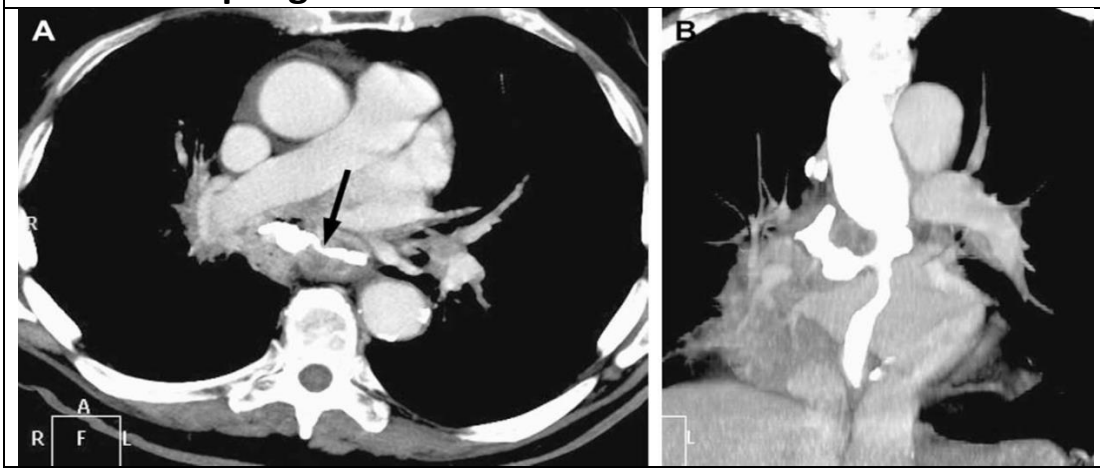
Signs associated with tracheal invasion are :-

- Evidence of extraluminal and endoluminal mass
- Destruction of the cartilage
- Tracheoesophageal or bronchoesophageal fistula

Direct tracheal invasion by thyroid carcinoma

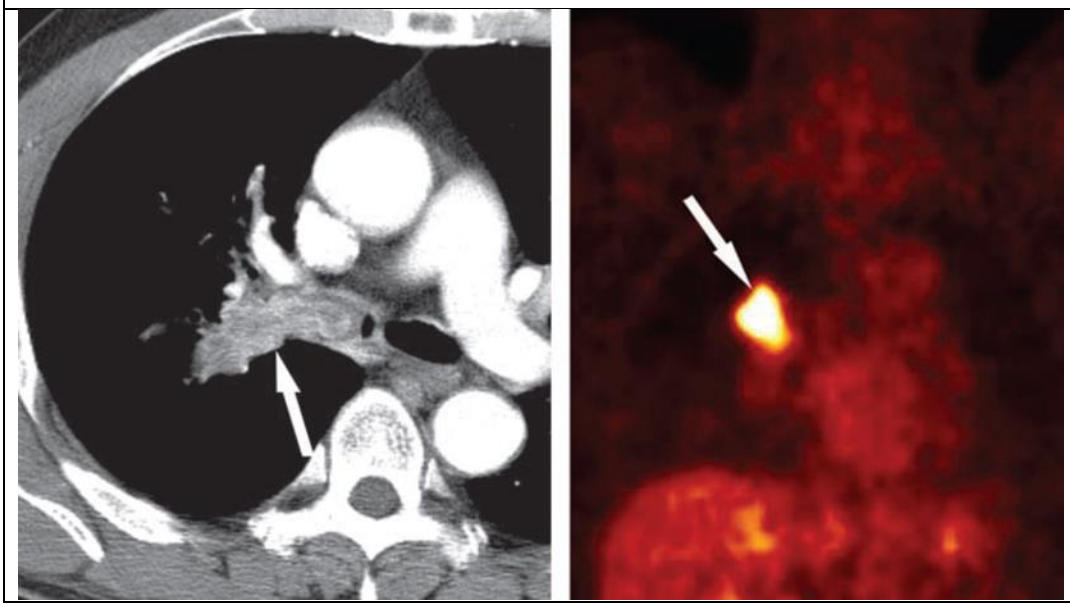


Esophageal cancer => Right bronchus invasion => Tracheoesophageal fistula



B) Tracheobronchial Metastasis

Breast,
Colorectal,
Renal,
Lung,
Ovarian,
Thyroid,
Uterine,
Testicular
Melanomas
Sarcomas



IV- Bronchiectasis

Bronchiectasis is permanent irreversible dilatation of the airways.

Clinical picture: Chronic productive cough

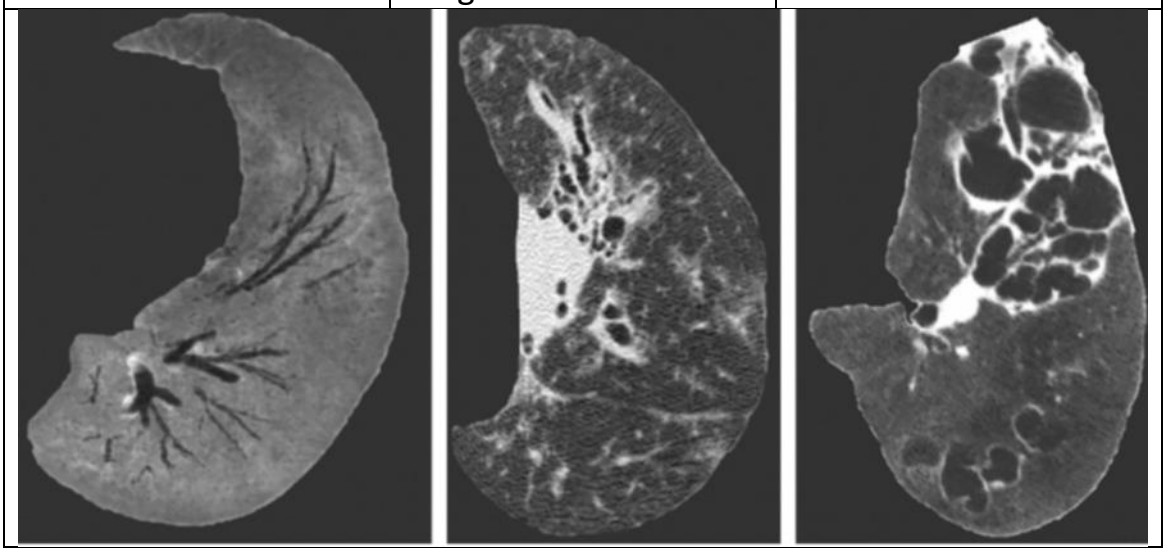
Radiological features:

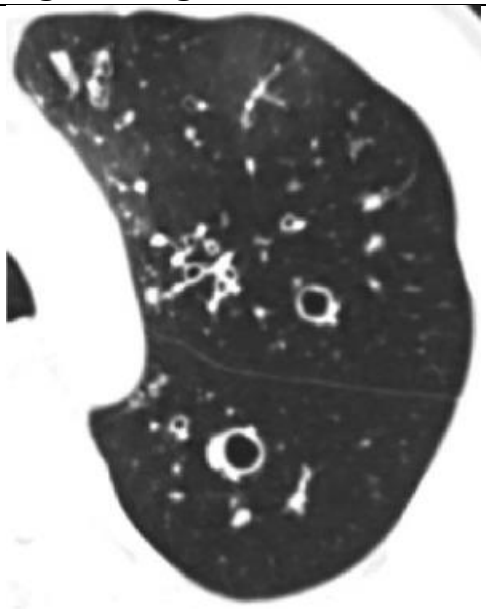
1) Lack of smooth progressive tapering of bronchi:
   - Normal airways not identified within 1 cm of the pleural
   - Bronchiectasis: bronchi seen in the outer 1/3 of the lung
2) Bronchial dilatation:

   Bronchial diameter (outer wall to outer wall) / Adjacent pulmonary a. diameter
   - Normal = 1:1
   - Mild bronchiectasis = 1-2 times
   - Moderate bronchiectasis = 2-3 times
   - Marked bronchiectasis > 3 times
3) Bronchial wall thickening:
   - Bronchial wall thickness ≥ Diameter of adjacent blood vessel
4) Longitudinal section => Tram-track appearance:
   - Horizontal parallel lines corresponding to the thickened dilated bronchi.
5) Transverse section => Signet ring sign:
   - Stone of ring = Pulmonary a. branch
   - Band of ring = thickened bronchial wall
6) 3 basic morphologic types: Cylindrical – Varicose - Cystic.
7) ± Mucus plugging
8) ± Air / fluid level
9) ± Air trapping => Mosaic attenuation
10) ± Tree-in-bud pattern

Complication:

1) Repeated pulmonary infections
2) Pulmonary fibrosis & Architecture distortion
3) Cor pulmonale and pulmonary arterial hypertension
4) Hemoptysis
5) Respiratory insufficiency
6) Aspergillus superinfection

A) Cylindrical bronchiectasis: Smooth tubular contour	B) Varicoid bronchiectasis: Tortuous beaded irregular contour	C) Cystic bronchiectasis: Saccular dilatation
		

Tram-track	**Signet ring**
	

Causes of Bronchiectasis

Cause	Distribution	Findings
Infection or aspiration		
Recurrent aspiration	Lower lung zone, peripheral	Bronchial wall thickening, aspirated material in bronchi, presence of hiatal hernia
Allergic bronchopulmonary aspergillosis	Upper lung zone, central	High-attenuation mucous plugging
Tuberculosis	Upper lung zone, focal or unilateral	Asymmetric upper lobe involvement, tree-in-bud nodules
Chronic atypical mycobacterial infection	Middle lobe and lingular	Most common in males with chronic obstructive pulmonary disease or in thin, older women; indolent symptoms
Swyer-James syndrome	Focal or unilateral	Bronchial wall thickening, increased lucency in the smaller lung due to airtrapping
Congenital conditions		
Cystic fibrosis	Upper lung zone, central	Extensive cystic and cylindrical bronchiectasis
Primary ciliary dyskinesia	Middle lobe and lingular	Situs inversus, chronic sinusitis, and bronchiectasis
Mounier-Kuhn syndrome	Central	Absence or atrophy of the elastic fibers and smooth muscle in tracheal and main bronchial wall
Williams-Campbell syndrome	Midorder bronchi	Cartilage deficiency of the midorder bronchi
Bronchial atresia	Focal or unilateral	Most common location
Fibrosing diffuse lung diseases		
Sarcoidosis	Upper lung zone, central	Bilateral hilar lymphadenopathy, perilymphatic nodules
Pulmonary fibrosis	Lower lung zone, peripheral	Varicoid bronchiectasis, architectural distortion, honeycombing in usual interstitial pneumonitis
Acute respiratory distress syndrome	Middle lobe and lingular	Varicoid bronchiectasis, diffuse ground-glass opacities
Endobronchial or peribronchial lesions		
Endobronchial tumor	Focal or unilateral	...
Foreign bodies	Focal or unilateral	...
Strictures	Focal or unilateral	...
Other		
Bronchiolitis obliterans	Diffuse	Significant cause of posttransplantation morbidity and mortality; diffuse airtrapping

A) Bronchiectasis with Upper lung zones predominance

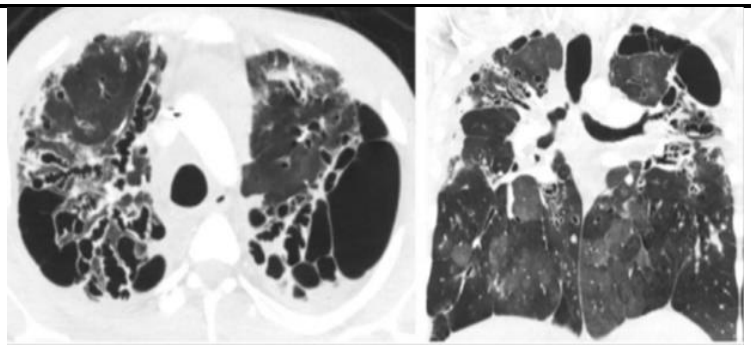
Cystic fibrosis – ABPA – Sarcoidosis – Silicosis – Tuberculosis.

1) Cystic Fibrosis:

Autosomal recessive disease

- ⇨ Abnormality in cystic fibrosis transmembrane conductance regulator protein
- ⇨ Abnormal chloride transportation across the cell membrane.
- ⇨ Decrease water content of the mucus
- ⇨ Thick tenacious mucus => Mucus plugging
- ⇨ Colonization of plugged airways by staph and pseudomonas
- ⇨ Recurrent pulmonary infections

- ❖ Abnormal sweat chloride test result
- ❖ Pulmonary:
  - o Pattern: cystic and cylindrical bronchiectasis
  - o Distribution: upper lung zones (Upper lobes + Apical segments of lower lobes)
- ❖ GIT:
  - o Pancreatic insufficiency: Steatorrhea – Malabsorption
  - o Meconium ilieus – Meconium peritonitis – Intussusception
- ❖ Sinusitis
- ❖ Infertility in males



2) Sarcoidosis:

- Pattern: Reticulo-nodular pulmonary infiltration
  - Nodules with lymphatic distribution: peribronchovascular, subpleural, interlobular septal
  - Fibrotic changes: reticular opacities, architectural distortion, traction bronchiectasis, bronchiolectasis, volume loss
- Distribution: Predominant upper- and middle-zone locations
- Bilateral hilar and mediastinal lymphadenopathy.

3) Silicosis

- Pattern:
  - Nodular pattern: Calcification 20%
  - Reticulo-nodular pattern
- Distribution: UL > LL
- Hilar lymphadenopathy: Egg shell calcification (5%)
- **Progressive massive fibrosis:**
  - UL posterior segment
  - Conglomerate masses formed by coalescent nodules
  - Vertical orientation
  - Fibrosis + Cavitation => Traction bronchiectasis

4) Post-Primary TB:

- Pattern:
  - Tree-in-bud nodules: suggest active endobronchial spread.
  - Exudative pattern: Patchy or confluent consolidation
  - Fibro-productive pattern:
    - Consolidation => TB Cavity ± Fungal ball (Aspergilloma)
    - Apical fibrosis => Traction bronchiectasis
- Distribution: Upper lung zones

5) Allergic Bronchopulmonary Aspergillosis (ABPA)

- Pattern: Finger in glove
  - Saccular bronchiectasis
  - Mucoid impaction
- Distribution: Upper lung zones

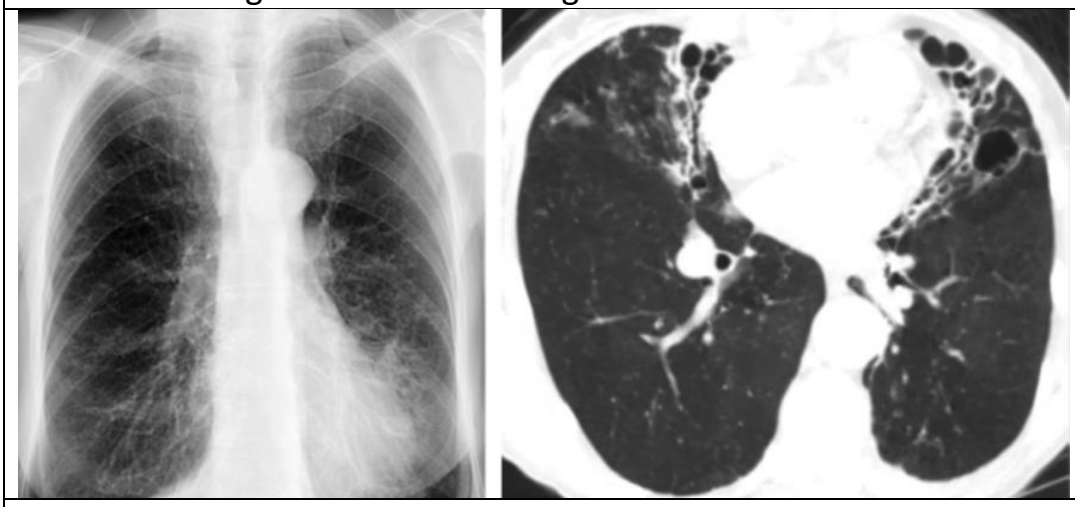
B) Bronchiectasis with Anterior Predominance

1) Atypical Mycobacterial Infection:

Pathogen: Mycobacterium avium intracellulare (MAI) and M. kansasii

- **Older females in good health with cough, low-grade fever, and weight loss, called Lady Windermere syndrome**
- **Older men with COPD**
- **AIDS patients**

Pattern: Bronchiectasis and tree-in-bud nodules
Distribution: Right middle lobe – Lingula.



2) ARDS

<u>Acute phase = Exudative phase (1-7 days):</u>

- Pattern: Consolidation / GGO
- Distribution: Bilateral patchy / multifocal / diffuse
- Gravity dependent
- No cardiomegaly - No pleural effusions - No Kerley A&B lines

<u>Late phase = Fibrotic phase (>15 days)</u>

Most radiographic pattern Resolve

- **<u>Pattern:</u>** Reticular interstitial disease => Fibrosis => Traction bronchiectasis
- **<u>Distribution:</u>** Anterior distribution (right middle lobe and lingula)
- **<u>Mechanism:</u>**
  - Parenchymal damage intrinsic to ARDS
  - Barotrauma resulting from prolonged mechanical ventilation
- The dependent portions of the lungs are protected from barotrauma by the typical gravitational gradient of consolidation in ARDS

C) Bronchiectasis with Lower Lung Zone Predominance

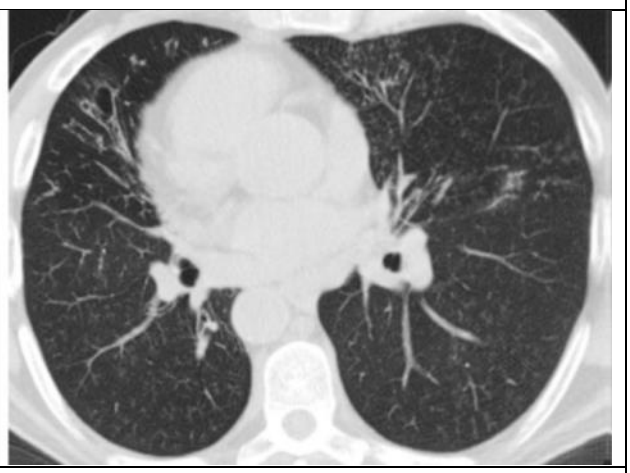
1) UIP = IPF:

- Age > 50y & M > F
- Insidious onset & progressive course of unexplained dyspnea on exertion & nonproductive cough.
- Duration of illness > 3 m
- Exclusion of other known causes of interstitial lung disease (eg, toxic effects of certain drugs, environmental exposures, and connective tissue diseases)
- Pattern:
    - Reticular intestinal pattern – Smooth septal thickening -
    - Traction bronchiectasis – Honey combing
- Distribution: Bilateral – Basal – Peripheral

2) Primary Ciliary Dyskinesia = Immotile Cilia Syndrome

An autosomal recessive disease => Impairing mucociliary clearance => repeated infections

Kartagener syndrome = Situs inversus + Chronic sinusitis + Bronchiectasis



3) α1-Antitrypsin Deficiency

Pttern: Panlobular emphysema – Chronic bronchitis – Bronchiectasis

Distribution: lower lung zones

4) Immunodeficiency

Bronchiectasis can occur in the context of congential or acquired immunodeficiency.

Repeated respiratory and gastrointestinal tract infections secondary to compromised serum and mucosal antibody protection.

5) Chronic Aspiration

Aspiration of gastric contents => direct damage to large and small airways => leads to airway inflammation.

Predisposing factors:

- Large hiatus hernia - GORD
- Patulous esophagus (including scleroderma)
- Esophageal motility disorders.
- Oropharyngeal dysphagia
- Neurologic disorders - Dementia - Bedridden state

Radiological findings:

Pattern:

- Centrilobular ground-glass nodules
- Tree-in-bud nodules
- Bronchiectasis

Distribution: bilateral peripheral lower lung zone (dependent)

D) Bronchiectasis with Central Predominance

1) Mounier-Kuhn Syndrome

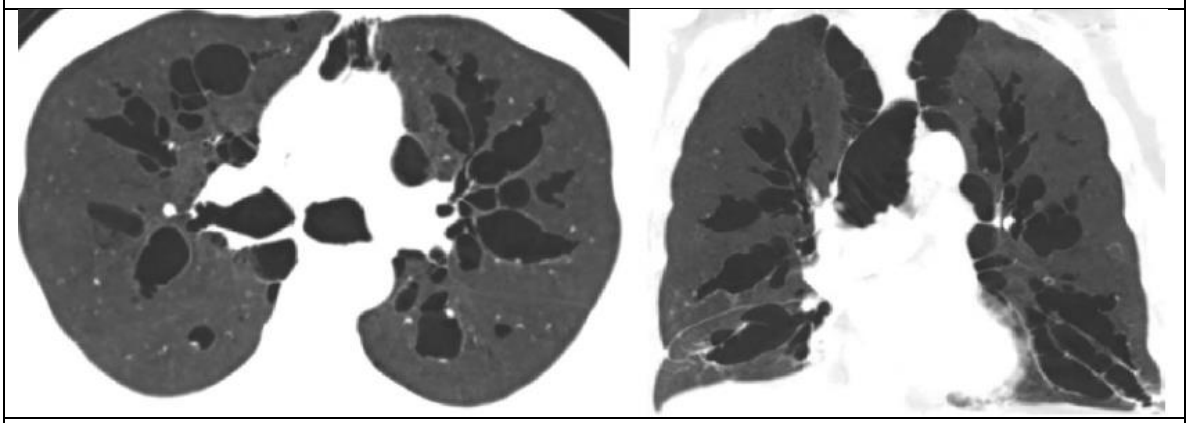
Congenital tracheobronchomegaly
Absence or marked atrophy of the elastic fibers and smooth muscle in the wall of the trachea and main bronchi

Dilatation of: Trachea + Main-stem bronchi + Segmental bronchi + Proximal subsegmental bronchi

Coronal tracheal diameter

> 25 mm in men

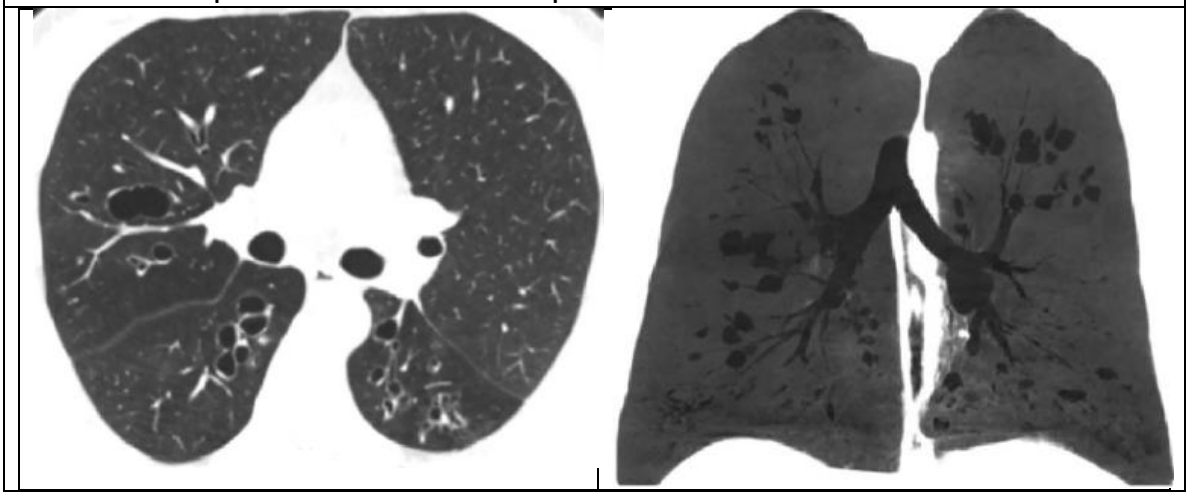
> 21 mm in women



2) Williams-Campbell Syndrome

A rare cartilage deficiency of the mid-order bronchi

Cystic bronchiectasis is limited to mid-order subsegmental airways
Trachea and proximal bronchi are spared

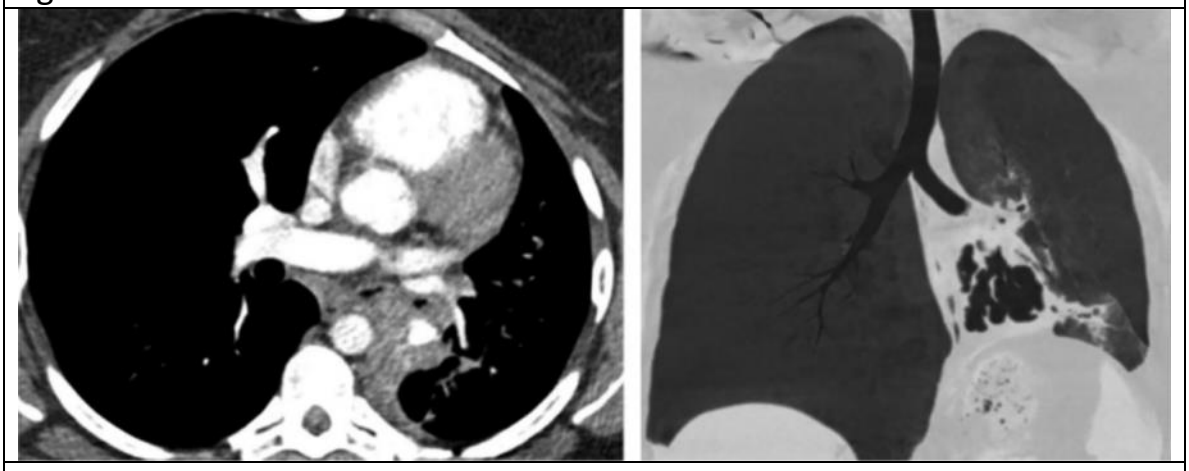


E) Focal Bronchiectasis

1) Endobronchial Tumor:

Chronic obstruction with a ball-valve mechanism can cause damage to the airway and superimposed postobstructive infection

Eg: Carcinoid tumor



2) Swyer-James Syndrome

Postinfectious bronchiolitis obliterans and usually occurs during childhood, Small lucent lung + Diffuse bronchiectasis.

F) Diffuse Bronchiectasis

Bronchiolitis Obliterans

BO is a major complication in long-term follow-up of patients after lung or heart-lung transplantation (Graft-versus-host disease)

Radiological findings:

- Mosaic attenuation with air trapping
- Bronchiectasis,
- Bronchial wall thickening

V- Chronic Obstructive Pulmonary Disease (COPD)

Two components:

- **Chronic bronchitis** is a clinical diagnosis: excessive mucus formation and cough for >3 months during 2 consecutive years.
- **Emphysema** is a pathologic diagnosis: abnormal enlargement of airspace distal to the terminal nonrespiratory bronchiole.

The exact etiology is unknown:

- Smoking
- Industrial air pollution
- Antitrypsin deficiency

Clinical Syndromes:

a) **Blue bloaters:**
   - Chronic bronchitis (Tussive) type of COPD
   - Episodic dyspnea due to exacerbation of bronchitis
   - Young patients

b) **Pink puffers:**
   - Emphysematous type of COPD
   - Progressive exertional dyspnea
   - Elderly patients

Radiological features of COPD:

A) Radiological features of chronic bronchitis:

Bronchial wall thickening:

- Longitudinal section => Parallel shadows
- Axial section => Thickening of bronchi imaged in.
- Increased lung markings (“dirty chest”)
- Accentuation of linear opacities throughout the lung
- Very subjective finding

B) Radiological features of Emphysema:

See chapter of emphysema

VI- Emphysema

Permanent abnormal enlargement + wall destruction of airspaces distal to the terminal bronchiole.

Patho-physiology:

- Smoking-related emphysema => Increase elastase production:
- Alpha-1-antitrypsin deficiency => Deficiency of neutralizing enzyme => Unopposed action of elastase:
  - ⇨ Elastase is powerful destructive enzyme
  - ⇨ Lung destruction and resultant emphysema.

Radiological Features:

CXR PA view => Hyperinflation:

- Prominent hilar vascular shadows
- Peripheral attenuation of vascular markings
- Low flat diaphragm: <1.5cm above straight line drawn between costo-phrenic and vertebero-phrenic junction
- Tenting of diaphragm
- Saber-sheath trachea: ↓ Coronal diameter of intra-thoracic trachea
  - AP diameter of the intrathoracic trachea (sagittal diameter) > lateral diameter (coronal diameter) = 2:1
- Ribbon shaped heart
- Barrel shaped chest
- Horizontal ribs + widely spaced ribs
- Increased lung lucency

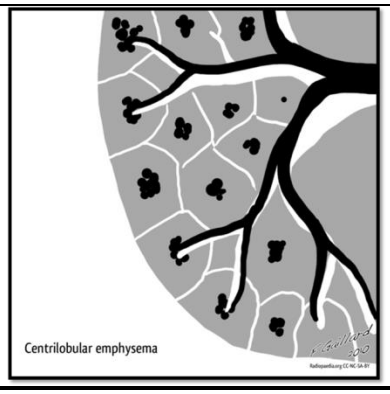
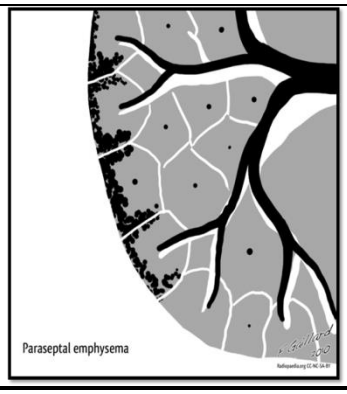
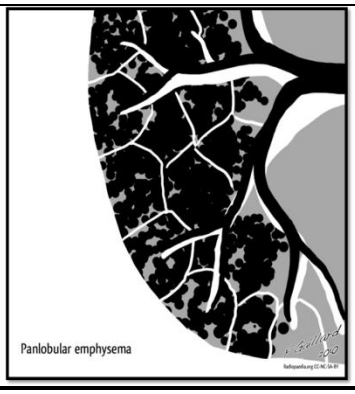
CXR Lateral view => Hyperinflation:

• ↑ Retrosternal space	• Anterior bowing of sternum
• ↑ Retrocardiac space	• Accentuated kyphosis

HRCT:

- Areas of decreased attenuation (Type, Distribution, Location)
- No visible walls
- Polygonal shaped 2ry pulmonary lobule
- Central dot sign (Central pulmonary artery)
- Peripheral pruning of pulmonary vessels
- Bulla: well defined thin walled air lucency > 1cm
- Bleb: < 1cm

Types of Emphysema

Centrilobular/ Centriacinar Emphysema	Paraseptal Emphysema	Panlobular / Panacinar Emphysema
Center of 2ry pulmonary lobule	Along periphery of lung and septal lines	Entire 2ry pulmonary lobule
Smoking related lung disease	seen in combination with other forms of emphysema	α1-Antitrypsin deficiency
UL		LL
		

1) Centrilobular Emphysema

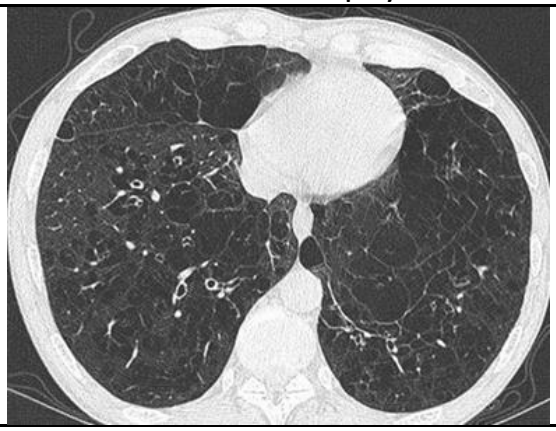
- **Centre of 2ry pulmonary lobule**
- **Smoking related lung disease:**
  - ⇨ RB-ILD => Centrilobular Nodules (UL)
  - ⇨ Centrilobular Emphysema => Centrilobular lucency (UL)
  - ⇨ PLCH => Bizarre shaped Cysts (UL)
  - ⇨ DIP => Diffuse ground glass opacities (Diffuse & Peripheral)
  - ⇨ UIP = IPF => Septal thickening + Honeycombing + Traction bronchiectasis (LL & Peripheral)

2) Paraseptal Emphysema

3) Panlobular / Panacinar Emphysema

- **Alpha-1-antitrypsin deficiency**
  - ⇨ Most common metabolic liver disease in children
  - ⇨ Deficiency of neutralizing enzyme
  - ⇨ Unopposed action of elastase
  - ⇨ Elastase is powerful destructive enzyme
  - ⇨ Lung destruction and resultant emphysema

Distribution: Bilateral LL
Pattern: Panlobular emphysema



4) Obstructive Emphysema

Inhalation of FB.
Rt > Lt. Entire lung / lobe / segment
Inspiratory film => Area of hyperlucency
Expiratory film

- ⇨ Air trapping
- ⇨ Shift of mediastinum to the contralateral side

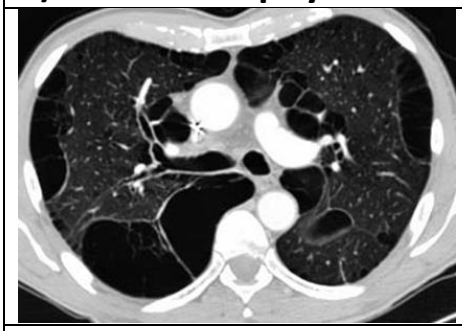
5) Compensatory Emphysema

Bnormal part of the lung => Collpase / Removed
Normal part of the lung => Hyperinflated & hyperlucent

6) Paracicatricical Emphysema

Distension & destruction of terminal airspaces adjacent to fibrosis.

7) Bullous Emphysema



8) Congenital Lobar Emphysema

Congenital overdestintion overdistention of a lobe
(NO alveolar destruction => Emphysema is misnomer)

Aetiology:

a) Idiopathic
b) Obstruction of airway (Valve mechnaism):

- Bronchial carticlage dysplasia - Brochial stenosis
- Insipissated mucus - Mucosal folds or webs
- Extensive vascular or mass compresssion

Distribution: LUL > RML > RUL

Radiological features:

- ⇨ 1st few days of life: Enlarged opaque hemithorax due to lung fluid trapped in the involved lobe
- ⇨ Gradually: fluid clears via vascular and lymphatic reabsorption
- ⇨ Classic appearance: Hyperinflated hyperlucent hemothorax

VII- Bronchial Asthma

Hyperirritability of airways causes reversible airway obstruction (bronchial smooth muscle contraction, mucosal edema, hypersecretion of bronchial secretory cells: bronchospasm).
The etiology is unknown (IgE participation).

Types

a) Extrinsic, allergic form

• Childhood asthma
• Immunologically mediated hypersensitivity to inhaled antigens

b) Intrinsic asthma

• Adults
• No immediate hypersensitivity

Radiographic Features

- **Normal CXR in majority of patients**
- **Severe or chronic asthma:**
  - Air trapping => Hyperinflation:
  - Bronchial wall thickening (tramlines): a nonspecific finding

Complications

- Acute pulmonary infection
- Mucus plugs
- Allergic bronchopulmonary aspergillosis (ABPA)
- Tracheal or bronchial obstruction
- Pneumomediastinum/pneumothorax

VII- Broncholithiasis

is a rare disorder of calcified/ossified material within the bronchial lumen.

Most common cause:

- **Erosion by and extrusion of an adjacent calcified granulomatous lymph node.**
  - ✓ TB – Histoplasmosis - Silicosis

Other causes of broncholithiasis include

- Aspiration of bone tissue or in situ calcification of aspirated foreign material
- Erosion by and extrusion of calcified or ossified bronchial cartilage plates
- Migration to a bronchus of calcified material from a distant site, e.g. pleural plaque or the from the kidney (nephrobronchial fistula)

Radiological findings:

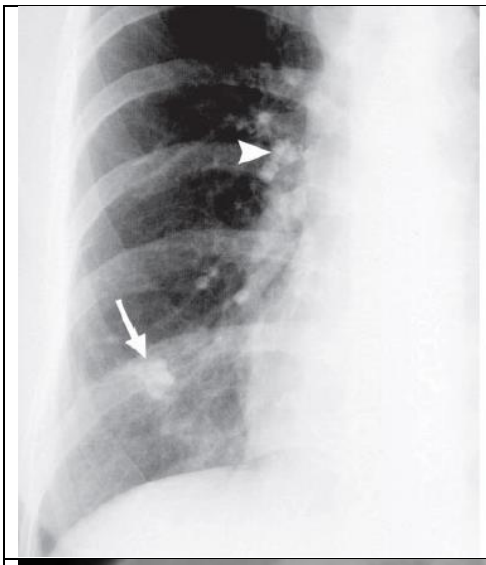
- Endobronchial calcified / ossified lesion

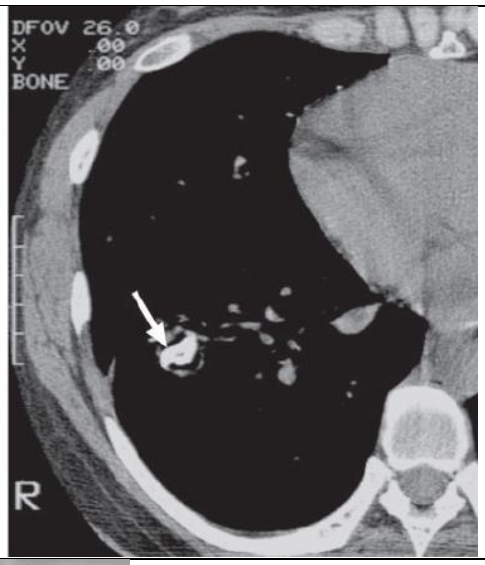
2ry effects: Segmental / Subsegmental:

- Atelectasis
- Consolidation
- Bronchiectasis
- Expiratory air-trapping
- Mucus plugging

Location:

Preferential sites are known to be the proximal right middle lobe bronchus (Middle Lobe Syndrome) and the origin of the anterior segmental bronchus of the upper lobes

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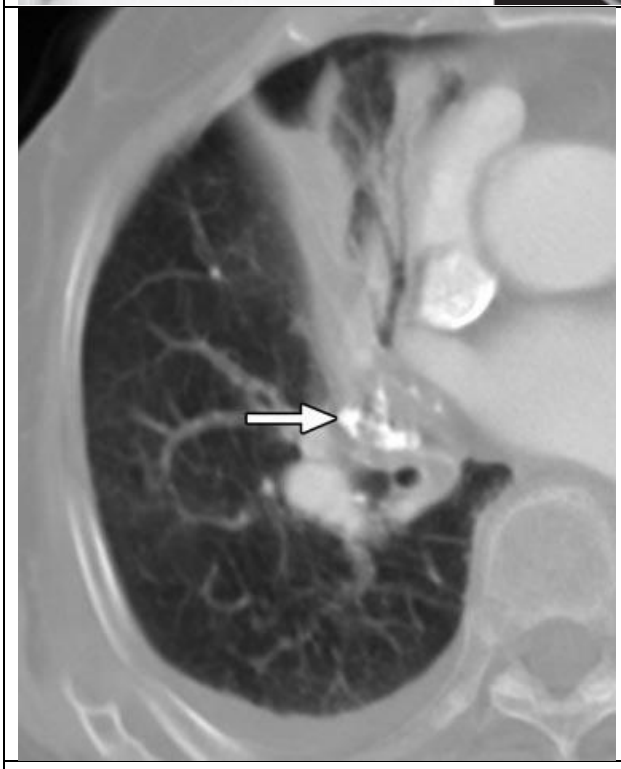


Small Airways Diseases

Small airways are defined as noncartilaginous airways with internal diameter < 2 mm
Located from 8th generation of airways down to terminal bronchioles (smallest airways without alveoli) and respiratory bronchioles (airways distal to terminal bronchioles that partake in gas exchange)

Classification of Small Airway Disease (Bronchiolar Disease = Bronchiolitis):

Primary bronchiolar disorders
Constrictive bronchiolitis
Acute bronchiolitis
Diffuse panbronchiolitis
Respiratory bronchiolitis
Mineral dust airways disease
Follicular bronchiolitis
Interstitial lung disease with prominent bronchiolar involvement
Hypersensitivity pneumonitis
Respiratory bronchiolitis–interstitial lung disease
Desquamative interstitial pneumonia
Organizing pneumonia
Bronchiolar involvement in large airways disease
Chronic bronchitis
Asthma
Bronchiectasis

Radiological Features:

- Centrilobular ground glass nodules
- Tree-in-bud pattern
- Mosaic attenuation: Areas of hyperlucency with small vessel caliber
- Air-trapping on expiratory CT
- Diffuse central and peripheral bronchiectasis (less common)
- Bronchial wall thickening

I- Bronchiolitis Obliterans

(Constrictive Bronchiolitis)

Aetiology:

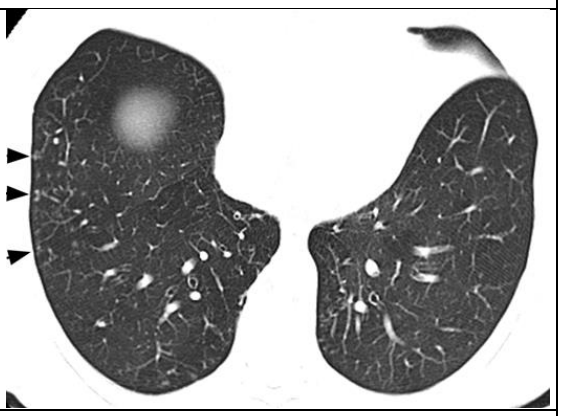
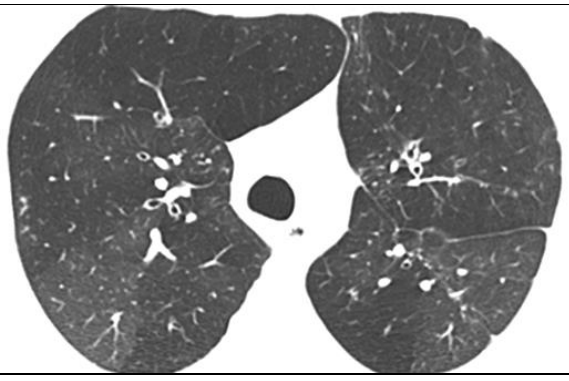
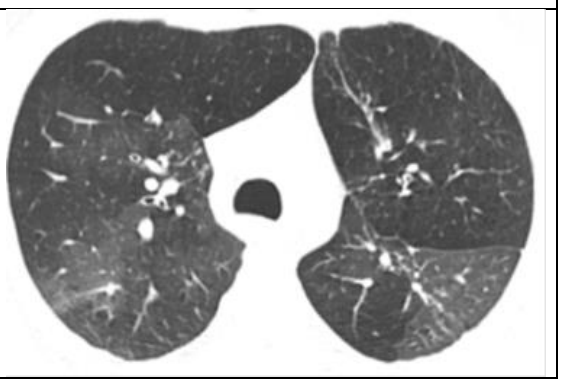
Most cases are idiopathic

- Postinfectious
  - Viral
  - Mycoplasma
- Collage vascular disease
  - Rheumatoid arthritis
  - Systemic lupus erythematosus
  - Systemic sclerosis
  - Mixed connective tissue disease
- Transplantation
  - Graft-versus-host disease
  - Allograft transplantation
- Drugs
  - D-penicillamine
  - Gold
  - Cocaine
  - Carmustine
- Cryptogenic
- Toxic fume exposure
  - Nitrogen dioxide
  - Sulfur dioxide
  - Ammonia
  - Chlorine
  - Phosgene
  - Diacetyl (popcorn workers)
- Miscellaneous
  - Ulcerative colitis
  - DIPNECH
  - Stevens-Johnson syndrome
  - Paraneoplastic pemphigus

II- Acute Infectious Bronchiolitis

Acute infections caused by:

- Bacterial
  - *Mycobacterium tuberculosis*
  - *M avium-intracellulare* complex
  - *Staphylococcus aureus*
  - *Haemophilus influenzae*
- Viral
  - Cytomegalovirus
  - Respiratory syncytial virus

Centrilobular nodules	**Tree-in-bud**
	
Inspiratory CT: Mosaic attenuation Low attenuation areas with small vessel caliber	**Expiratory CT:** Air-trapping Accentuation of low attenuation areas
	

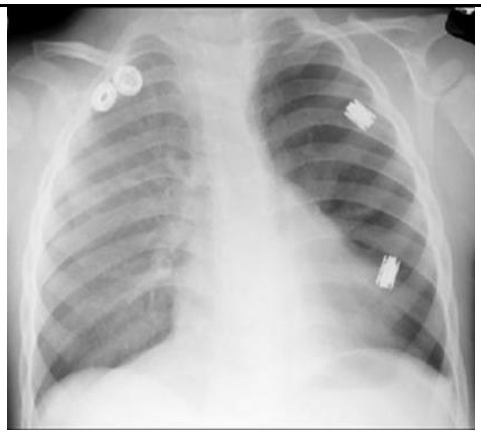
❖ Swyer-James Syndrome / Macleod Syndrome

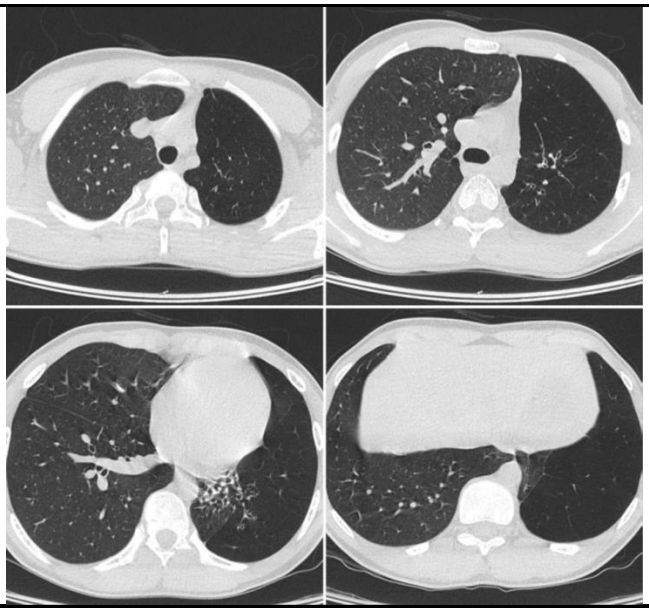
An acquired hypoplastic lung that develops following severe obliterative bronchiolitis, leading to bronchiolar obstruction, bronchiectasis, and distal airspace destruction

Typically follows a viral respiratory infection such as adenoviruses or Mycoplasma pneumoniae infection in infancy or childhood

Imaging Findings:

- Unilateral small hyperlucent lung.
- Unilateral small hilum
- Normal or reduced volume during inspiration.
- Mosaic attenuation: Areas of hyperlucency with small vessel caliber
- Air trapping during expiration.
- ± Bronchiectasis





Bronchogenic Carcinoma

The most common cause of deaths worldwide

Risk factors:

- Smoking accounts for 85% of all bronchogenic carcinoma
- Occupational and environmental exposures: Radon, asbestos, radiation exposure, uranium

4 major pathologic cell types:

- Small cell carcinoma
- Non small cell lung carcinoma.
    - Squamous cell carcinoma
    - Adenocarcinoma
    - Large cell carcinoma.

Adenocarcinoma	**35%**	**peripheral invasion of the overlying pleura , may cause bronchial obstruction by submucosal infiltration**
Squamous cell carcinoma	**30%**	**central , invasion of the main stem bronchus , pulmonary parenchyma and lymph nodes**
Small cell lung carcinoma	**20%**	**Central with rapid growth , Stronger liklihood for metastases Greater response to chemo and radiotherapy .**
Large cell carcinoma	**15%**	**Peripheral rapid invasion of the parenchyma early metastases.**

Clinical Manifestations:

- Bronchopulmonary : chest symptoms , lung abscess
- Pleural: malignant effusion, chylous, serous or empyema.
- Mediastinal (mass, nodes).

Extra thoracic Manifestations :

- SVC syndrome
- Distant metastases.
- Paraneoplastic syndrome: mostly with SCLC. ACTH production , PTH like production & hypercalcemia, hypertrophic osteoarthropathy , cerebellar ataxia , subacute cerebellar atrophy

Radiological Features:

I. Characterization of the primary lesion:
- Solitary pulmonary nodule / mass
- Malignant Cavity.
- Consolidation
- Unilateral hilar mass.
- Superior sulcus tumor.

II. Post-obstructive:
- Atelectasis
- Consolidation
- Bronchiectasis

III. Local relationship to:
- Pleura
- Chest wall
- Mediastinum
- Airways and vascular structures

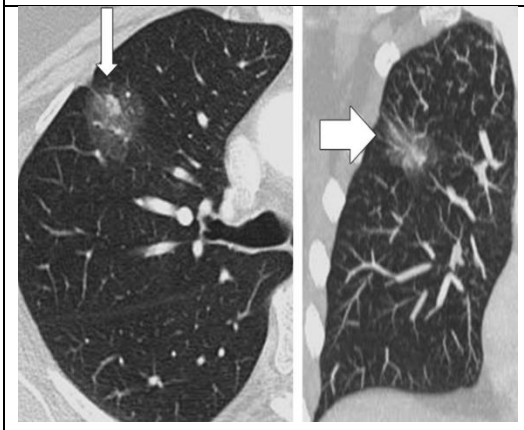
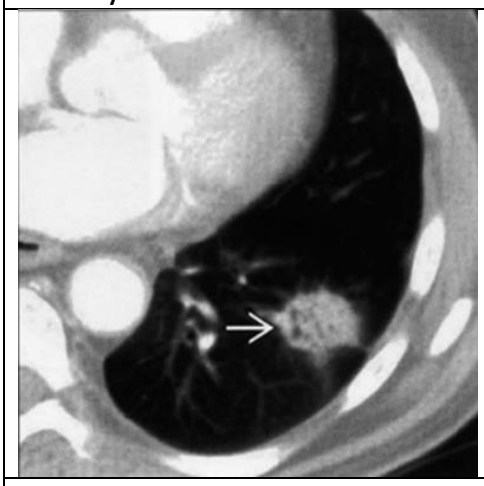
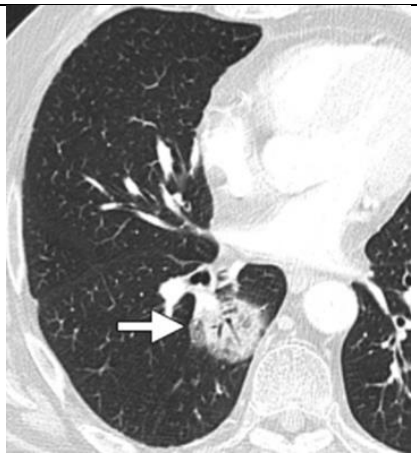
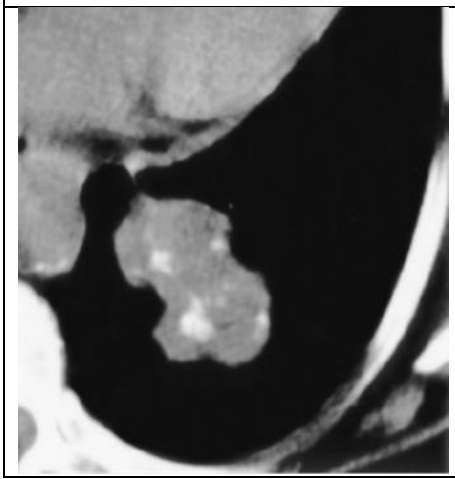
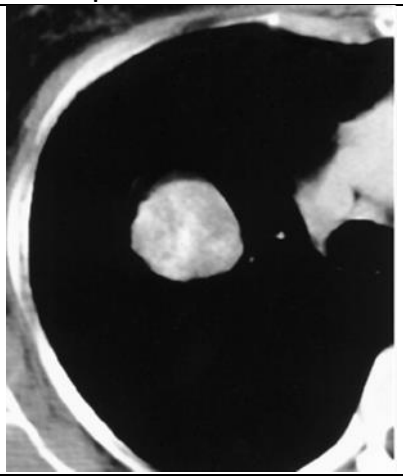
IV. **Distant tumor spread**
- to bones, liver, brain and suprarenal glands

I- Radiological Features of Primary Tumor

A) Solitary Pulmonary Nodule / Mass:

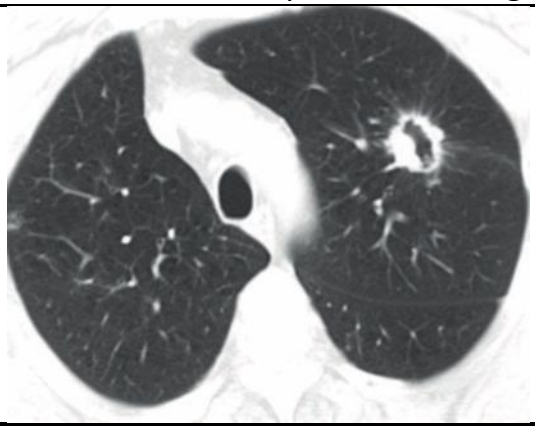
Features suggesting malignancy:

1) Size: Nodules approaching 3 cm, more likely to be malignant.
2) Attenuation:
   - Ground glass attenuation nodule (GGAN) => 34% malignant
   - Partly solid (Mixed solid and ground glass attenuation) => 50%malignant
   - Solid Nodule => 7% malignant
3) Margins:
   - Spiculated margins (sunburst or corona radiata sign)
   - Lobular or irregular contour
4) Calcifications: Punctate, eccentric, and amorphous calcifications
5) Cavitation: Thick – irregular wall
6) Pseudocavitation=Bubbly Lucencies: Small focal low attenuation within nodule
7) Air Bronchogram Sign
8) Halo Sign: denoting tumor invasion.
9) Growth rate:
   - Malignant solid nodules => volume doubling time 20 –400 days
   - 2-Year rule: No change in 2 years implies benignity.
10) Enhancement:
   - Malignant nodules enhance >20 HU
   - Benign nodules enhance <15 HU
11) Metabolism:
   - SUV cutoff of 2.5 has been used to differentiate between benign and malignant nodules

Partially solid nodule	Spiculated margins = Corona radiate signs
	
Pseudocavitation = Bubbly Lucencies	Air bronchogram sign
	
Punctate calcification	Amorphous calcification
	

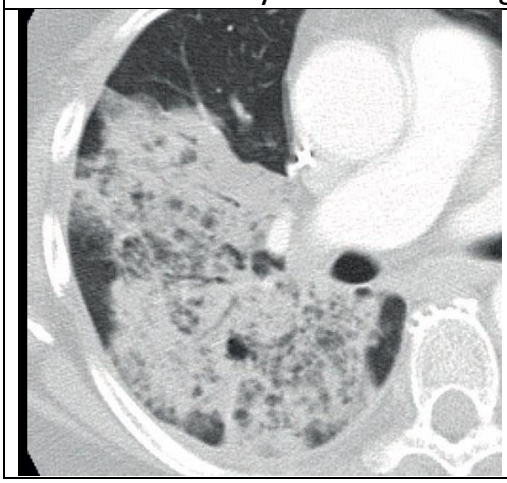
B) Malignant Cavity:

- Thick wall > 15mm
- Inner surface: Irregular / Nodular / Solid mural component
- Outer Surface: Speculated margins



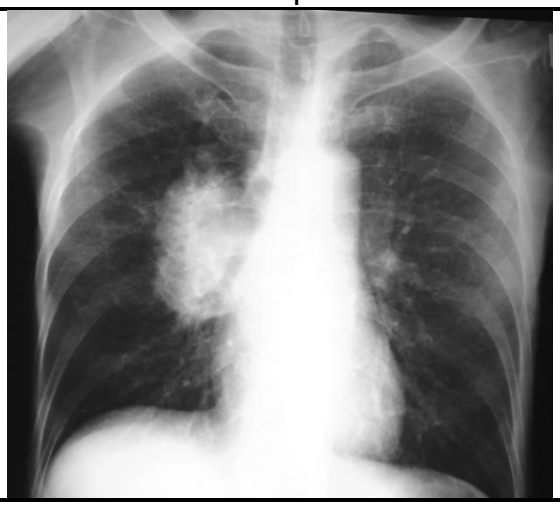
C) Consolidation:

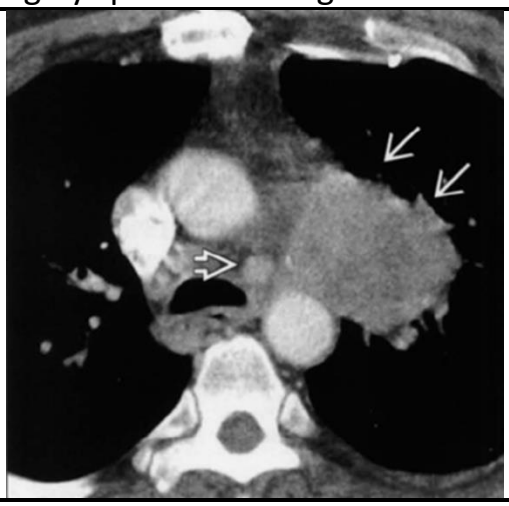
- Patch of consolidation with air-bronchogram within
- Consolidation that is indistinguishable from pneumonia can be seen with BAC (invasive mucinous adenocarcinoma).
- BAC is usually a non-resolving consolidation with normal WBC count.



D) Unilateral Hilar Mass

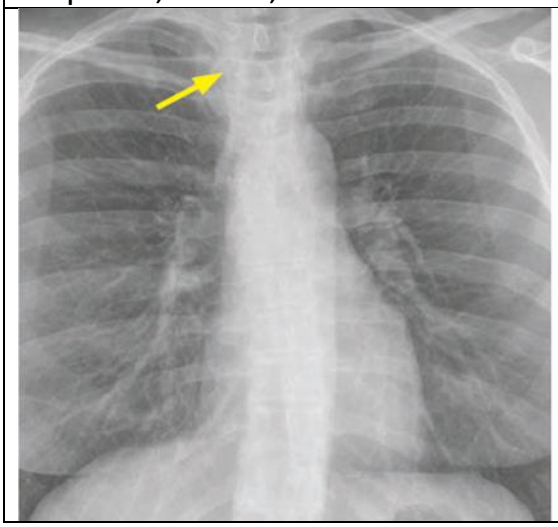
- A hilar mass is a common presentation of squamous cell and small cell carcinoma.
- Hilar enlargement may be due to a primary central tumor or nodal metastasis from a parenchymal neoplasm.
- Tumor may compress and narrow the bronchus.
- A tapered bronchus is highly specific for lung cancer

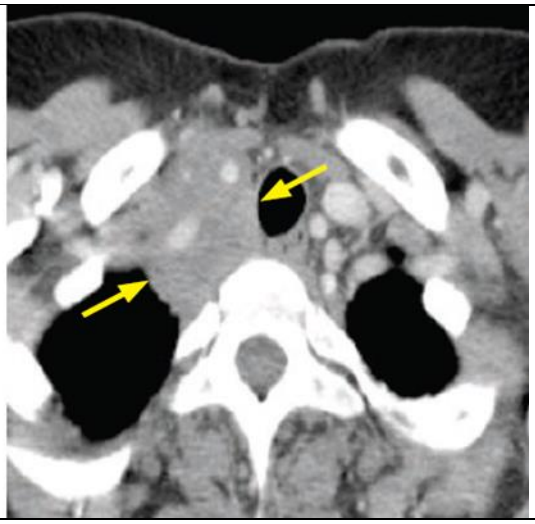




E) Superior Sulcus Tumor / Pancoast Tumor

- A superior sulcus tumor is a lung cancer occurring in the lung apex.
- A Pancoast tumor is a type of superior sulcus tumor with involvement of the sympathetic ganglia causing a Horner syndrome, with ipsilateral ptosis, miosis, and anhidrosis.

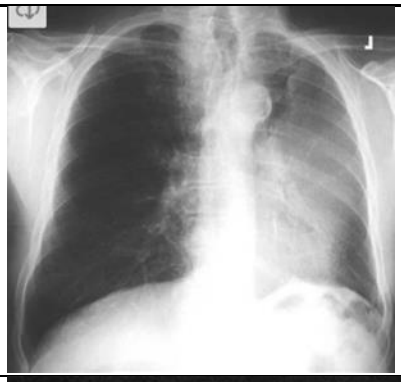
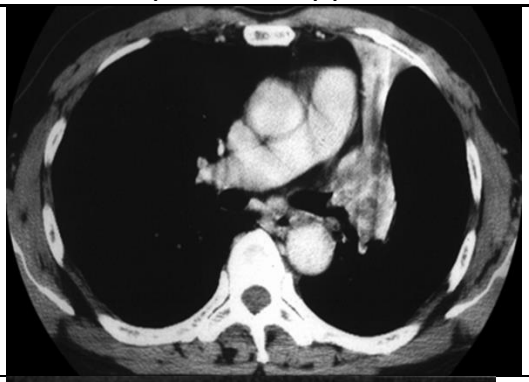
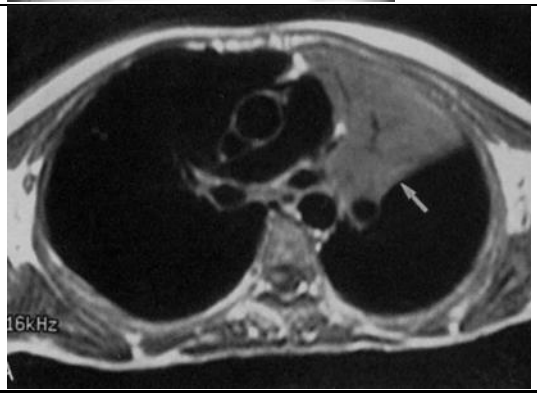




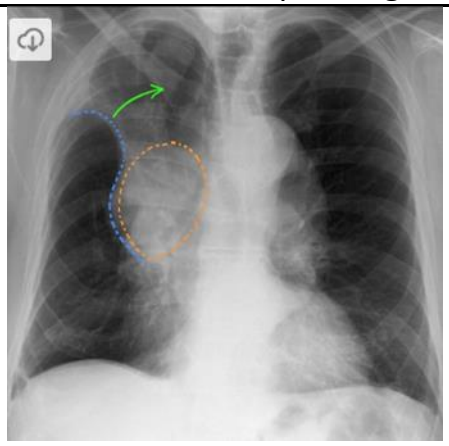
II. Obstructive Effect:

- Bronchial obstruction by the primary tumor causing obstructive pneumonia and atelectasis of the lung distal to the tumour.
- Depending on the location of the tumour, segmental or lobar collapse.

- Luftsichel sign = Left upper lobe collapse +
- Hyperinflation of the superior segment of the left lower lobe interposing itself between the mediastinum and the collapsed left upper lobe

- Golden S-sign: Right upper lobar collapse +
- Central mass expanding the hilum

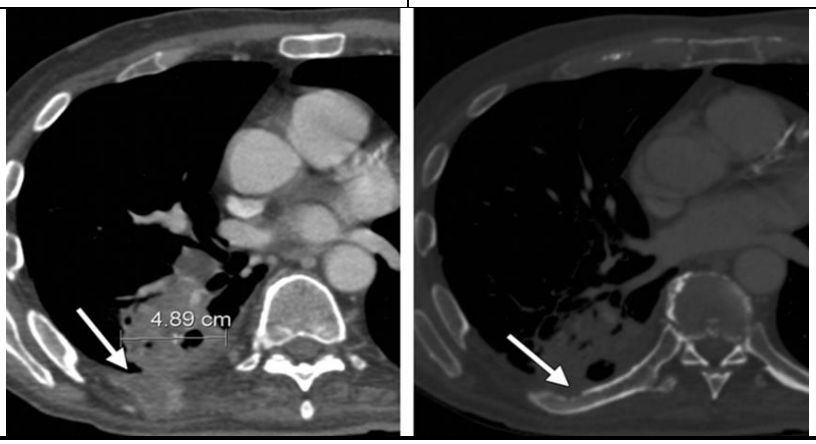


III- Tumor Extent:

A) Chest Wall Invasion => Stage T3:

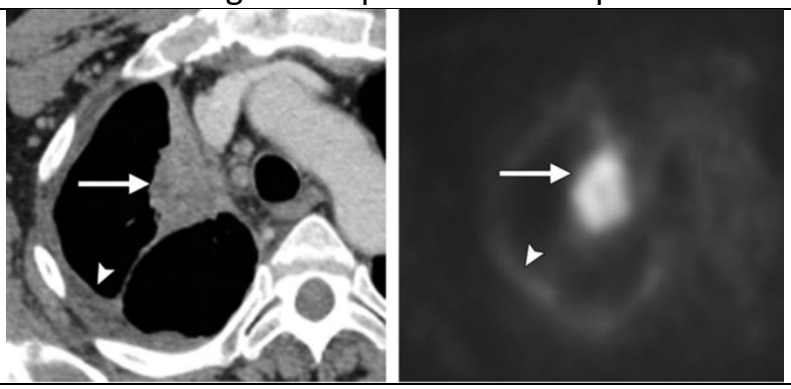
- Rib destruction
- Chest wall mass
- Obtuse angle at the point of contact between the tumor & the pleura
- Ratio between the diameter of the tumor and the length of tumor contact with the pleura.
- Fat plane between the tumor and chest wall

Chest wall mass	Rib erosion



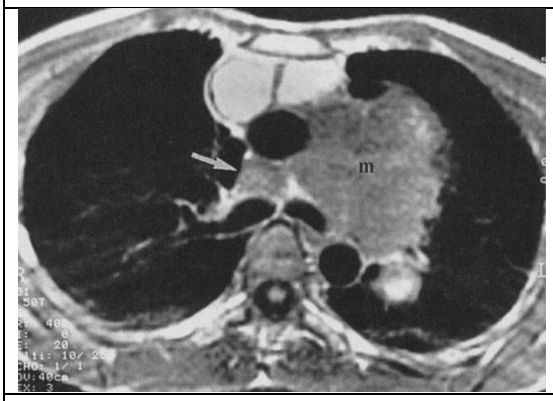
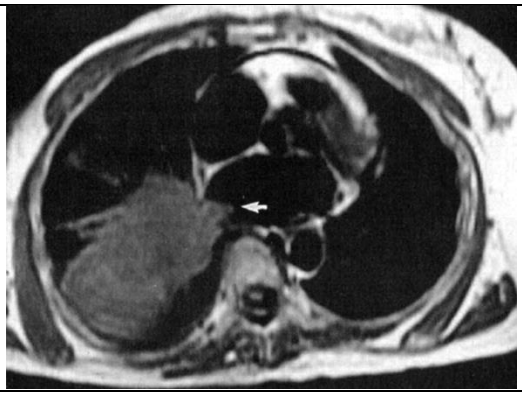
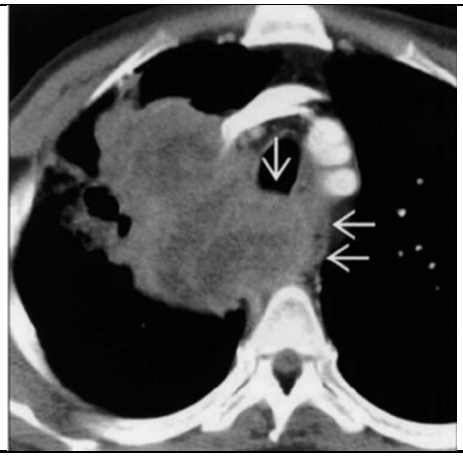
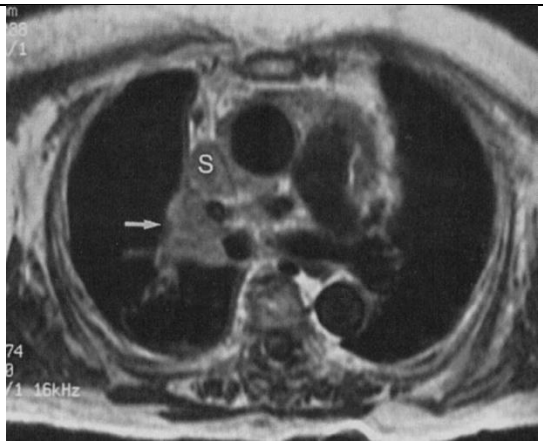
B) Pleural-Pericardial Invasion => Stage M1a

- Pericardial effusion with enhancing nodules is highly suggestive of malignant involvement of the pericardium.
- Nodular and enhancing pleural thickening is suggestive of metastatic pleural disease.
- Both CT and MR imaging findings are inconclusive for the determination of benign versus malignant pleural and pericardial disease.
- Diagnostic thoracentesis is still essential in evaluating for the presence of malignant cells in lung cancer patients with a pleural effusion



C) Mediastinal Invasion => Stage T4:

- Minimal invasion= tumor within the mediastinal fat = may be amenable for surgical resection
- Gross invasion = involvement of vital structures= precludes surgical resection
- Encasement , compression , displacement or actual invasion of the great vessels, pericardium, heart, trachea, bronchi and eosophagus
- Predictors of aortic invasion: > 90^{o}contact with aortic wall.

Mediastinal fat invasion	Lt Atrium Invasion
	
SVC occlusion	
	

International Association for the Study of Lung Cancer (IASLC) recommended 7th TNM Staging 2009 of the American Joint Committee on Cancer (AJCC) for small cell and non-small cell lung cancer staging:

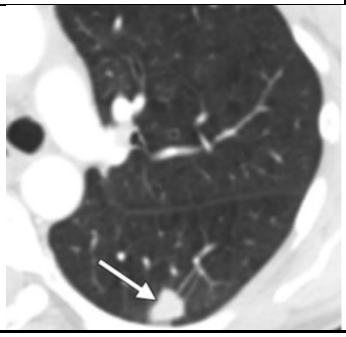
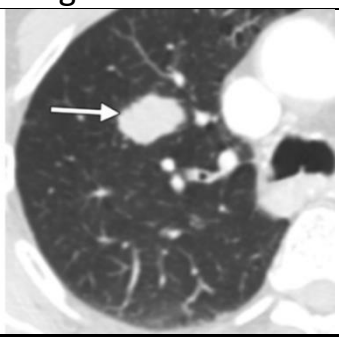
Stage	T	N	M
IA	Up to T1b	N0	M0
IB	T2a	N0	M0
IIA	Up to T2a (or T2b if N0)	N1	M0
IIB	T2b (or T3 if N0)	N1	M0
IIIA	Up to T3 (or T4 if N0–1)	N2	M0
IIIB	Any T (or T4 if N2)	N3	M0
IV	Any T	Any N	**M1a** or **M1b**

Staging-based Treatment Regimens

- **Early-stage disease** (stage IA, IB, IIA, IIB, and, in some cases, IIIA disease)
  - ⇨ Surgically resectable
  - ⇨ Possible role for neoadjuvant or adjuvant chemotherapy and radiation therapy.
- **Stage IIIB disease:**
  - ⇨ Surgical inoperable
  - ⇨ Chemotherapy–radiation therapy becomes the primary treatment .
- **Stage IV Metastatic disease**
  - ⇨ Surgical inoperable
  - ⇨ Main exceptions: Solitary adrenal or brain metastatic focus.

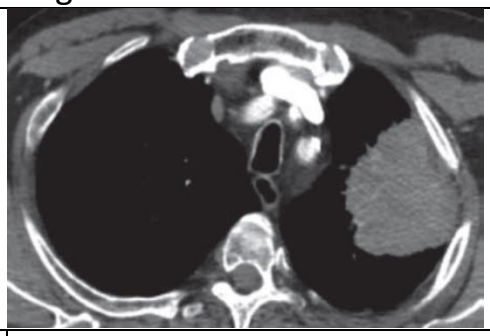
Stage T1

Tumors surrounded by lung or visceral pleura
Endobronchial lesions without invasion proximal to a lobar bronchus.

Stage T1a= Lesion < 2cm	Stage T1b= Lesion 2-3cm
	

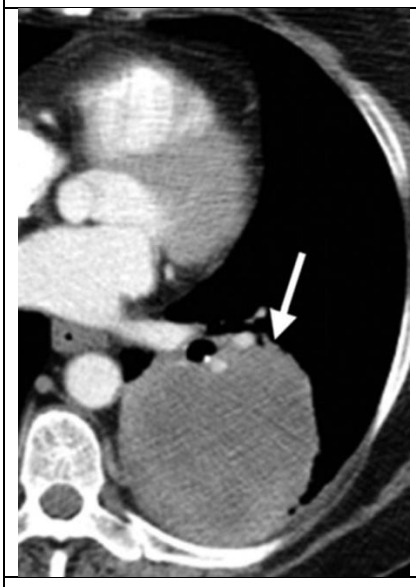
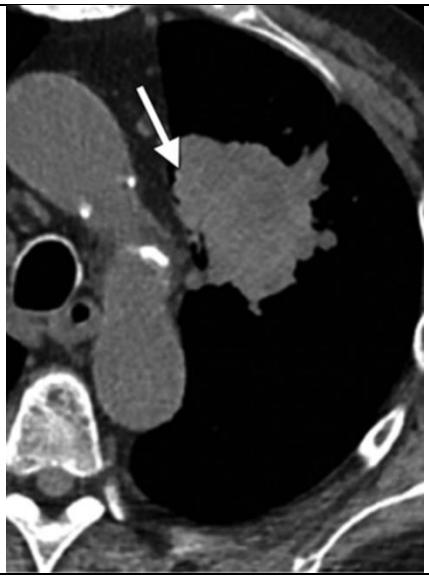
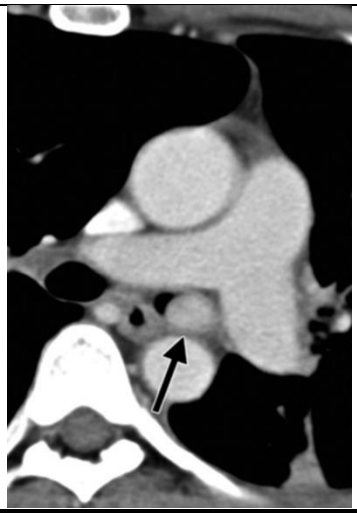
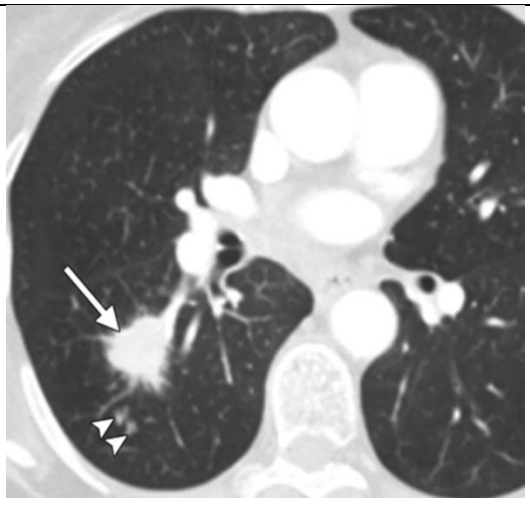
Stage T2

T2a: Tumor > 3 cm but ≤ 5 cm. T2b: Tumor > 5 cm but ≤ 7 cm.
Tumors with local invasion of the visceral pleura.
Atelectasis and obstructive pneumonitis extending to the hilar region but not involving the entire lung.
Endobronchial lesions > 2 cm distal to the carina

Stage T2a Tumor = 3-5 cm	Stage T2b Tumor = 5-7 cm
	
Endobronchial lesion > 2 cm from the carina	Centrally located lung nodule (arrow) causing atelectasis or postobstructive pneumonia that does not involve the entire lung

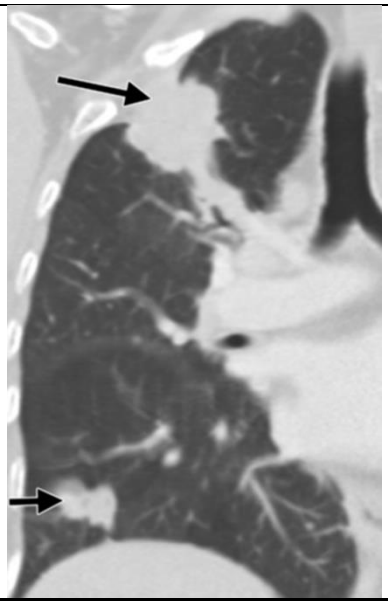
Stage T3

Tumors > 7 cm.
Separate tumor nodules in the same lobe as the primary lesion.
Endobronchial lesions < 2 cm distal to the carina
Tumors with local invasion of the chest wall, diaphragm, mediastinal pleura, and parietal pericardium.
Superior sulcus tumors
Tumors with atelectasis and obstructive pneumonitis of the entire lung.

Tumor > 7cm	Tumor extension to Mediastinal Pleura
	
Endobronchial mass (arrow) <2 cm from the carina	Satellite nodules in the same lobe
	

Stage T4

- Tumors of any size that demonstrate local **invasion of mediastinum** or carina, trachea, heart, great vessels, esophagus, or vertebral bodies.
- Separate tumor nodules in the same lung but not in the same lobe as the primary lesion.

Satellite nodule in another lobe in the same lung	Stage T4: Tumor extension to mediastinum / carina Stage M1a: Pleural effusion + thickening
	

N Staging

Supraclavicular zone

1 Low cervical, supraclavicular, and sternal notch nodes

SUPERIOR MEDIASTINAL NODES

Upper zone

2R Upper Paratracheal (right)

2L Upper Paratracheal (left)

3a Prevascular

3p Retrotracheal

4R Lower Paratracheal (right)

4L Lower Paratracheal (left)

AORTIC NODES

AP zone

5 Subaortic

6 Para-aortic (ascending aorta or phrenic)

INFERIOR MEDIASTINAL NODES

Subcarinal zone

7 Subcarinal

Lower zone

8 Paraesophageal (below carina)

9 Pulmonary ligament

N1 NODES

Hilar/Interlobar zone

10 Hilar

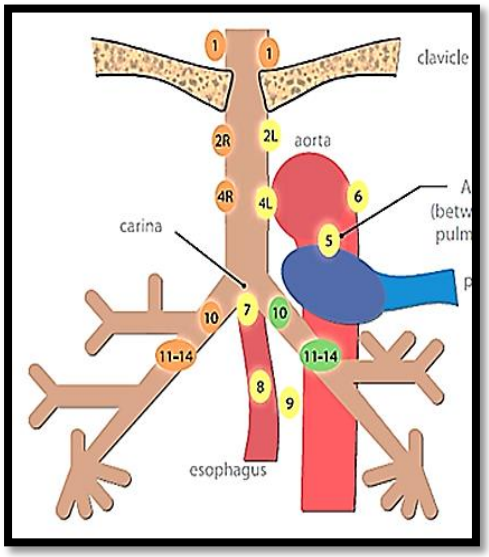
11 Interlobar

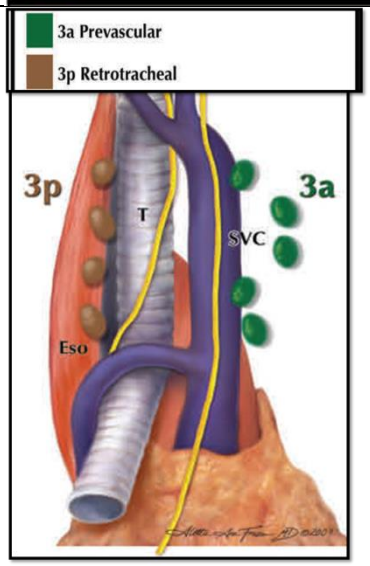
Peripheral zone

12 Lobar

13 Segmental

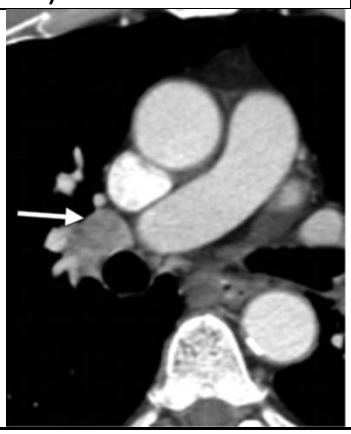
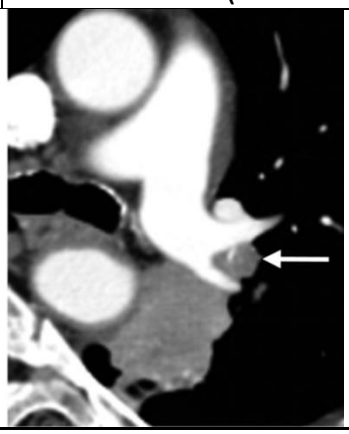
14 Subsegmental



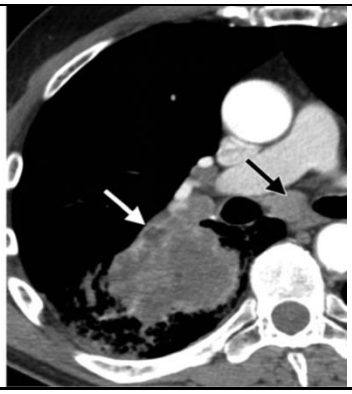


- N0: No lymph node metastases.
- N1: Ipsilateral hilar or intrapulmonary lymph nodes.
- N2: Ipsilateral mediastinal nodes.
- N3: Contralateral lymph nodes or supraclavicular nodes on either side.

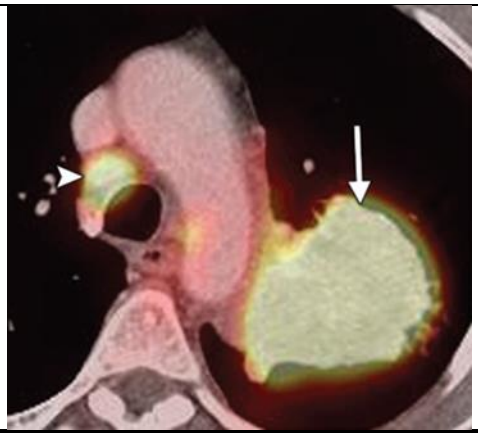
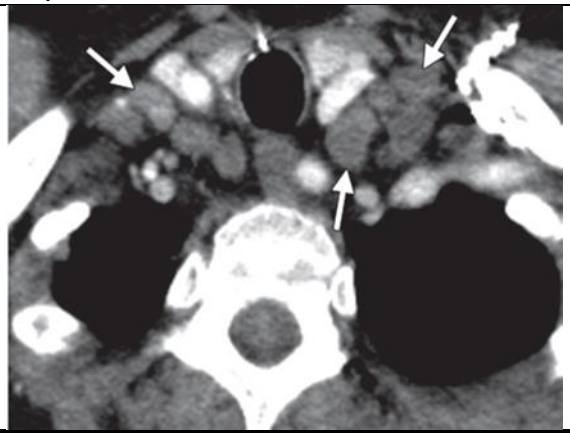
Stage N1: Hilar, interlobar, lobar, segmental, and subsegmental LNs

Hilar LN (level 10)	Interlobar LN (level 11)
	

Stage N2: Ipsilateral Mediastinum LNs

Right upper para-tracheal LN (Level 2)	Right lower para-tracheal LN (Level 4)	Subcarinal LN
		

Stage N3 = Stage IIIB = Inoperable

Contralateral Mediastinal LN	Supraclavicular LNs
	

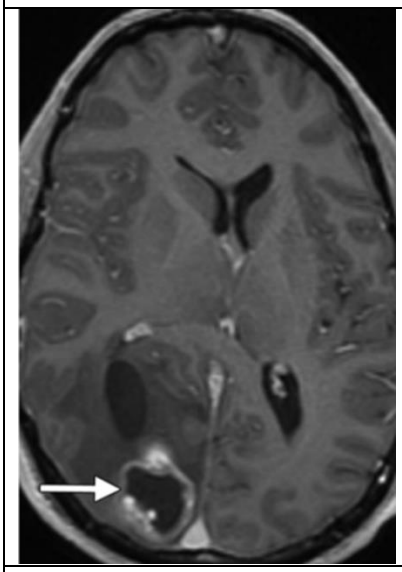
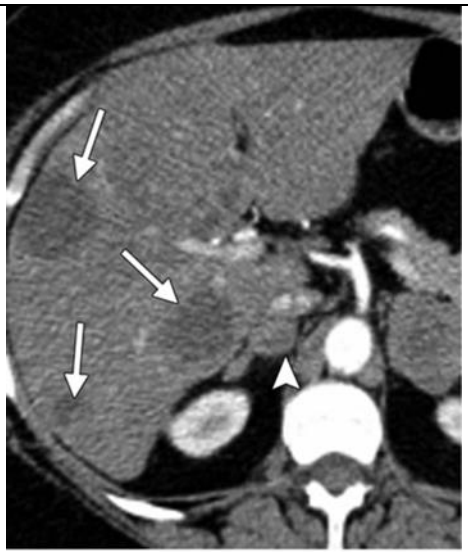
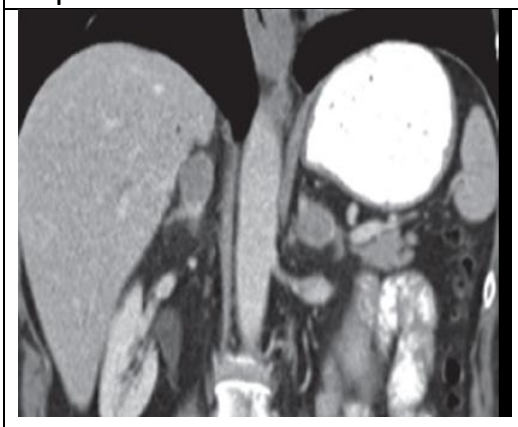
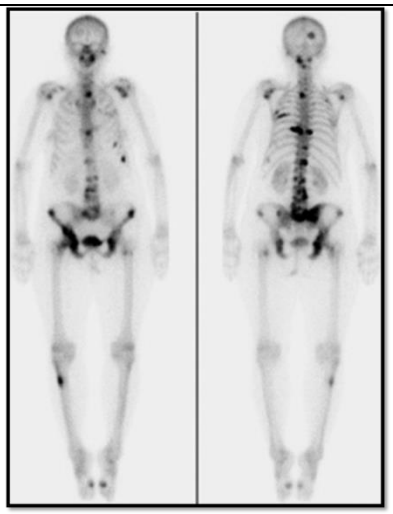
M Stage => Stage IV Disease

Stage M1a = Intrathoracic Metastasis

- Malignant pleural effusion
- Pleural dissemination
- Pericardial disease
- Pulmonary nodules in the contralateral lung

Stage M1b = Extrathoracic Metastasis

Spread to the liver, adrenal gland, brain, bone, and other locations away from the chest

Brain Metastasis	Liver Metastasis
	
Suprarenal Metastasis	Bone Metastasis
	

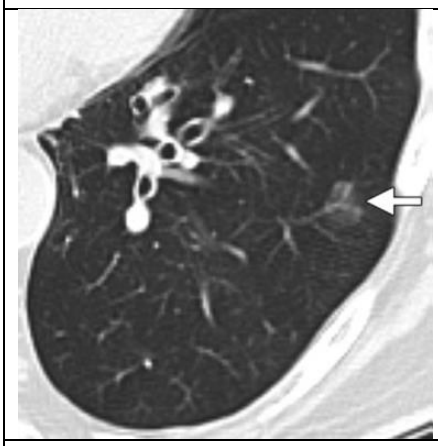
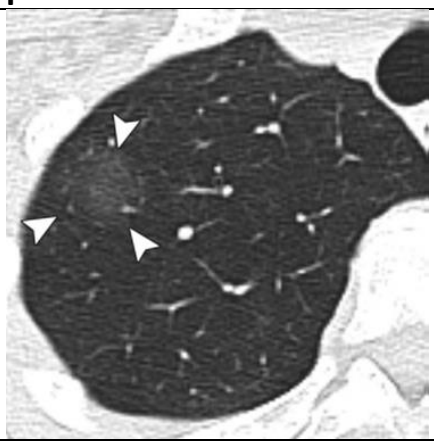
Adenocarcinoma

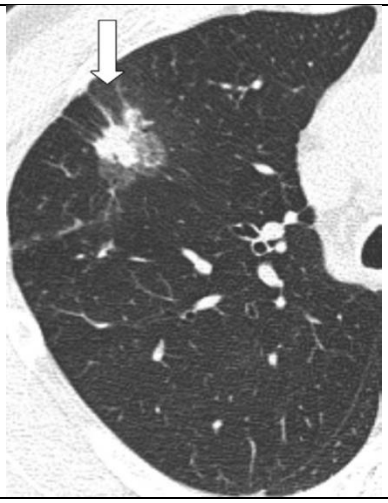
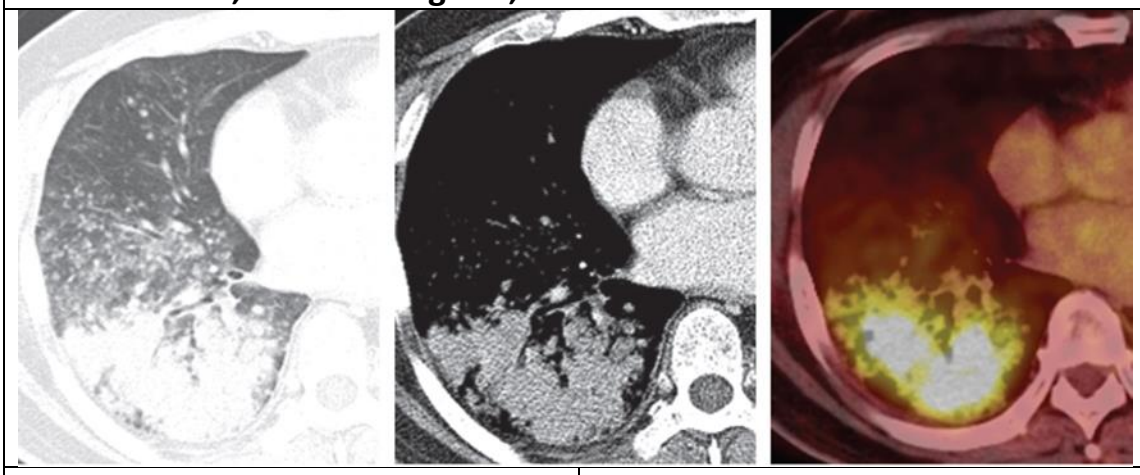
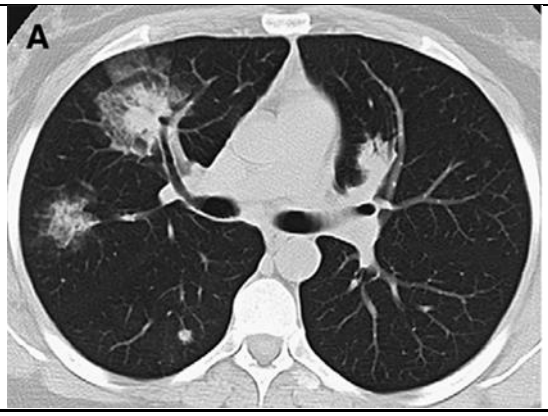
Previously Bronchioloalveolar carcinoma

New Classification of Adenocarcinoma 2011

Table 3: Classification of Nonmucinous Forms of Lung Adenocarcinoma and CT Features of Subsolid Nodules

2011 IASLC, ATS, and ERS Classification	CT Features
Atypical adenomatous hyperplasia	GGAN
Adenocarcinoma in situ	GGAN with a possible solid component
Minimally invasive adenocarcinoma	GGAN, partly solid nodule
Lepidic-predominant adenocarcinoma	Partly solid nodule, solid nodule
Invasive adenocarcinoma classified by predominant subtype	Partly solid nodule with a solid component, solid nodule

Atypical Adenomatous Hyperplasia (AAH) **Pure ground-glass attenuation nodule (GGAN) < 1 cm**	**Adenocarcinoma in situ (AIS)** **Pure ground glass attenuation nodule (GGAN) < 3cm** **Pure lipidic growth** **No stromal, vascular, lymphatic or pleural invasion**
	
Minimally invasive adenocarcinoma (MIA): **Subsolid nodule <3cm** **Predominantly nonsolid nodule** **No vascular, lymphatic, or pleural invasion**	**Lepidic predominant adenocarcinoma (LPA) (Nonmucinous BAC)** **Subsolid nodule <3cm** **Necrosis may be present** **Invasion of lymphatics, vessels and**

Solid component < 5 mm (arrows) **Stromal invasion < 5mm**	**pleura** **Solid portion of the nodule >5mm** **Stromal invasion > 5 mm**
	
Invasive mucinous adenocarcinoma (Mucinous BAC)	
Consolidation, air bronchogram; mass like	
	
Multifocal/Multilobar consolidation	**Solid and subsolid nodule**
	

Bronchopulmonary Neuroendocrine Tumors

4 Types:

e. Carcinoid Type1 (Typical)
f. Carcinoid Type 2 (Atypical)
g. Large cell neuroendocrine tumors (LCNE)
h. Small cell carcinoma (SCC)

WHO Classification:

D. Well-differentiated tumors = Typical Carcinoid tumors = Low-grade
E. Well-differentiated tumors = Atypical carcinoid tumors = Intermediate grade = more aggressive + metastases.
F. Poorly differentiated tumors = Small cell carcinoma + Large cell carcinoma = high-grade malignancies with a poor prognosis

Location:

GIT carcinoid (85%)

- Appendiceal carcinoid - 50% of all carcinoid tumours
- Small bowel carcinoid
    - Terminal ileum ~ 90% of all small bowel carcinoid tumours
- Gastric carcinoid

Bronchopulmonary carcinoid (20-30%):

- Central bronchial carcinoid (Main, lobar or segmental bronchi) (60%)
- Peripheral pulmonary carcinoid tumors (40%)
- Tracheal location: very rare

Primary hepatic carcinoid
Ovarian carcinoid (0.5% of carcinoid tumours)
Thymic carcinoid

Associations:

Sporadic but may be associated with:

- **Multiple endocrine neoplasia (MEN) type 1**
- **Zollinger Ellison syndrome**
- **Neurofibromatosis type I**

1) Bronchopulmonary Carcinoid Tumors

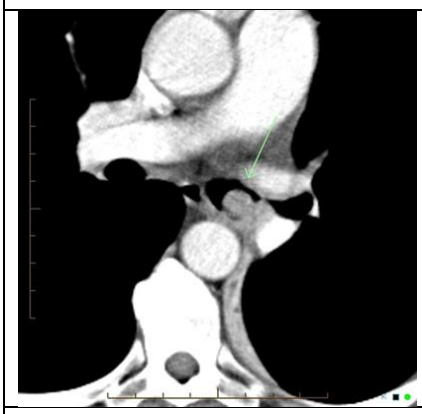
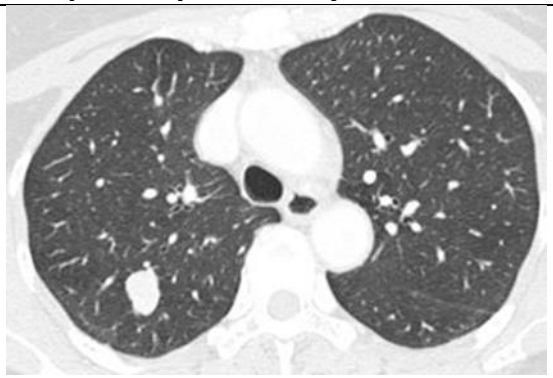
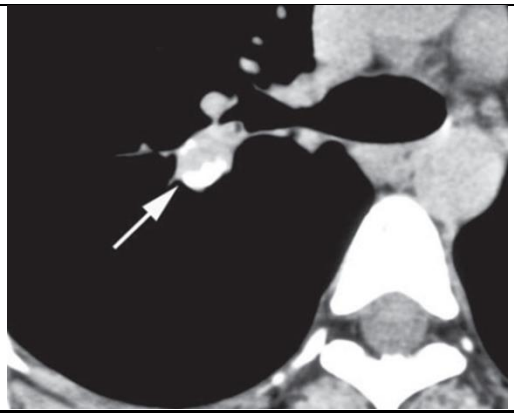
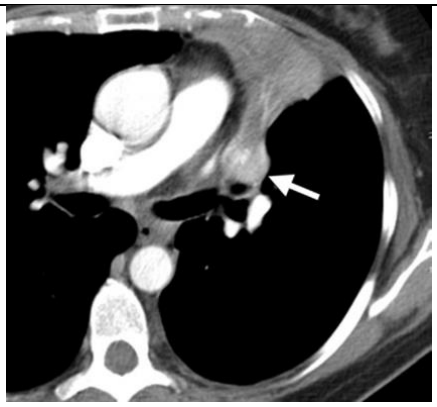
- Most frequent primary pulmonary neoplasm in childhood
- Age: Middle age
- Sex: M = F
- Typical (Type I) > Atypical (Type 2)
- Central location > Peripheral location

Location:

- Central: bronchial carcinoid (Main, lobar or segmental bronchi) (60%)
- Peripheral: pulmonary carcinoid tumors (40%)
- Tracheal location: very rare

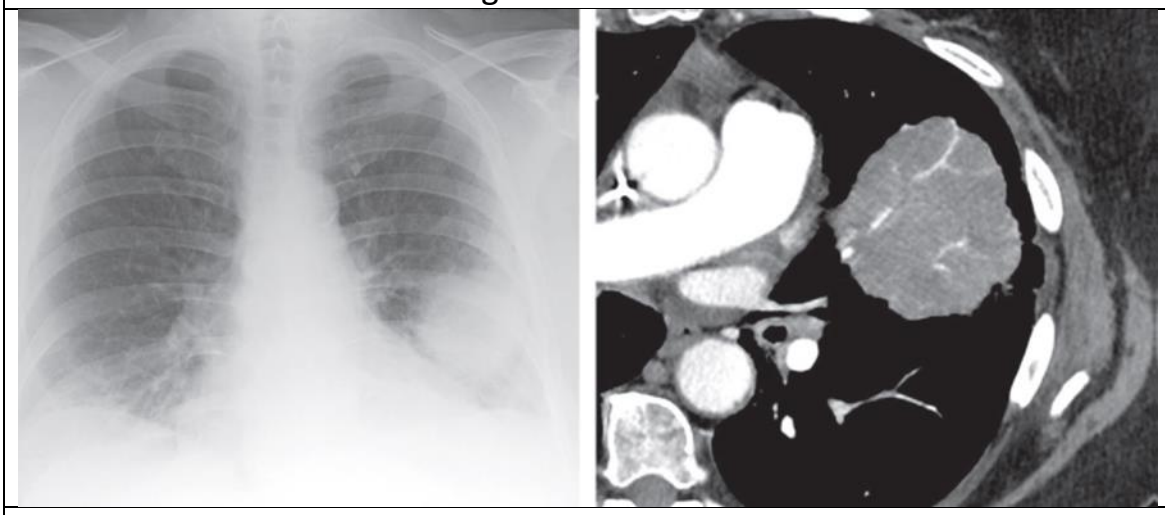
Radiological Features:

- A hilar or perihilar mass which is usually well-defined, round or ovoid.
- Small tumors may be entirely intrabronchial
- Marked homogeneous contrast enhancement due to high vascularity.
- Calcification (usually eccentric)
- Distal atelectasis / pneumonia

Endobronchial lesions	Peripheral pulmonary nodule
	
Eccenteric Calcification	**Distal atelectasis**
	

2) Large Cell Neuroendocrine Cancer

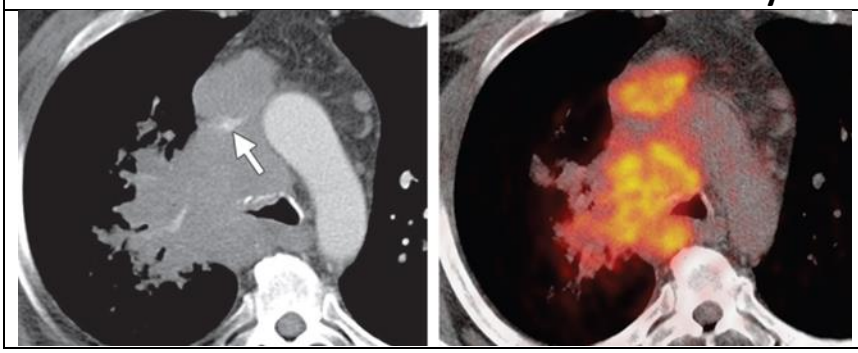
- High grade neuroendocrine neoplasm.
- Old Men Heavy smoker
- Large Peripheral pulmonary mass
- Well defined lobulated margins



3) Small Cell Lung Carcinoma (SCLC)

- Most common neuroendocrine lung neoplasm.
- 15%–20% of lung malignancies.
- Old Men Smokers
- Most aggressive type
- Worse Prognosis
- Rapid growth rate
- Large central mediastinal or hilar mass/nodal complex
- Invasion of mediastinal structures
- Pleuro-pericardial invasion
- 60-70% metastatic

Central mass with mediastinal invasion => SVC Syndrome



❖ SCLC Staging

Historically:

Veterans Administration Lung Cancer Study Group (VALSG) staging system divided SCLC into:

Limited stage SCLC (LS-SCLC) and Extensive stage SCLC (ES-SCLC)

Table 2: Modified VALSG Staging System

LS-SCLC
Confined to a single radiation port
Ipsilateral mediastinal or supraclavicular lymph nodes
Contralateral mediastinal or supraclavicular lymph nodes
Ipsilateral pleural effusions (benign or malignant)
ES-SCLC
Not confined to a single radiation port
Metastatic disease

NEW:

The International Association for the Study of Lung Cancer (IASLC) has recommended that the current 7th edition of the American Joint Committee on Cancer (AJCC) TNM staging system for lung cancer replace modified VALSG staging system

Pulmonary Lymphoma

HD (80%) > NHL (20%)
Nodal (90%) > Extra-nodal (10%)
Bimodal age distribution = 30y & 70Y

Clinical Picture:

- "B" symptoms are present in 40%
    - Unexplained weight loss – Fever – Night sweats
- A large mediastinal mass may produce:
    - Chest pain, cough or shortness of breath

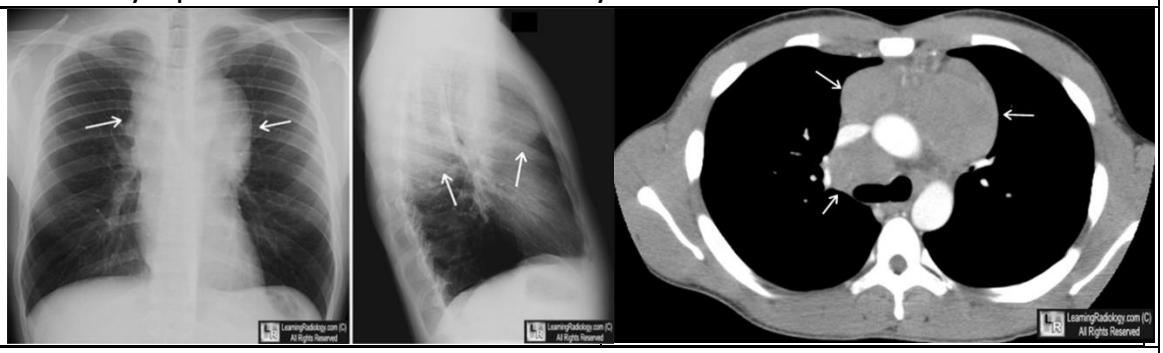
Staging (Ann Arbor)

Stage I limited to one lymph node bearing group
Stage II two or more non-contiguous groups on the same side of the diaphragm
Stage III lymph node groups on both sides of the diaphragm
Stage IV extranodal involvement-such as lung or brain

Other modifiers
S – Splenic involvement
B – Presence of B symptoms (temperature >38°C, drenching night sweats, unexplained loss of >10% of body weight in the preceding 6 months)
A – Absence of B symptoms
X – Presence of bulky disease
E – Contiguous involvement of extranodal sites

1) Hilar or Mediastinal Adenopathy (95%):

- Most common manifestation
- Anterior mediastinal /retrosternal + Paratracheal LNs commonly involved
- Confined to anterior mediastinum in 40%
- Hilar lymph nodes involved bilaterally in 50%



2) Bronchovascular form:

- Most common type of involvement
- Coarse reticulonodular pattern

3) Subpleural form:

- Circumscribed subpleural masses

4) Pneumonic Form:

- Diffuse nonsegmental infiltrate
- Massive lobar infiltrates (30%)
- Homogeneous confluent infiltrates with shaggy borders
- Contain air bronchogram

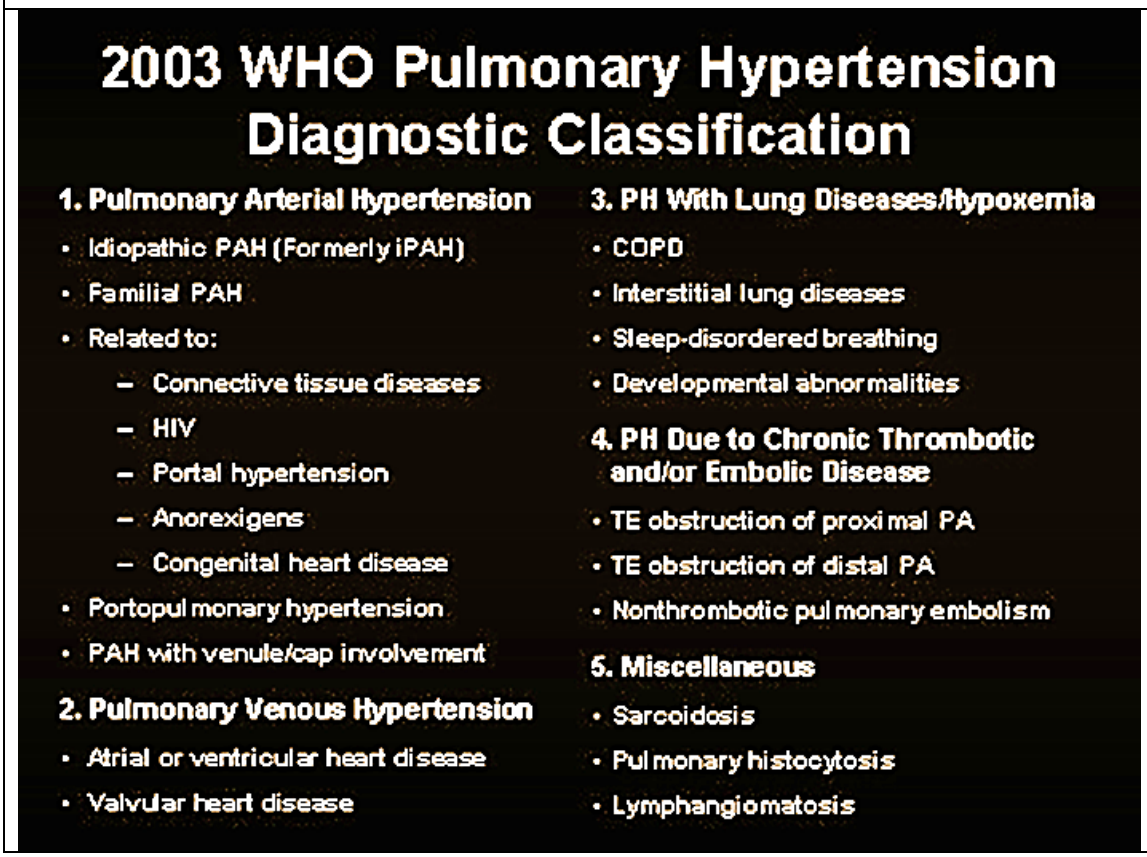
5) Nodular Form:

- Multiple nodules <1 cm in diameter

6) Pleural Effusion:

- Effusion usually does not contain malignant cells

Pulmonary Hypertension

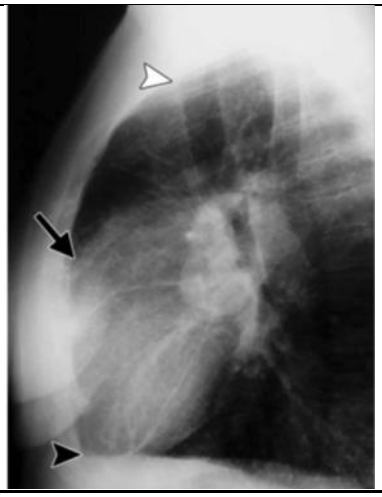
Abnormal elevation of pressure in pulmonary circulation Mean pulmonary arterial pressure > 25 mmHg Pulmonary capillary wedge pressure ≥18 mm Hg
Clinically, the term pulmonary hypertension is used to encompass both pulmonary arterial and pulmonary venous hypertension


CXR: PA view

- Prominent main pulmonary artery (arrow)
- Dilated right interlobar artery (arrowhead)
- Pruning of peripheral pulmonary vascularity.

CXR: Lateral view

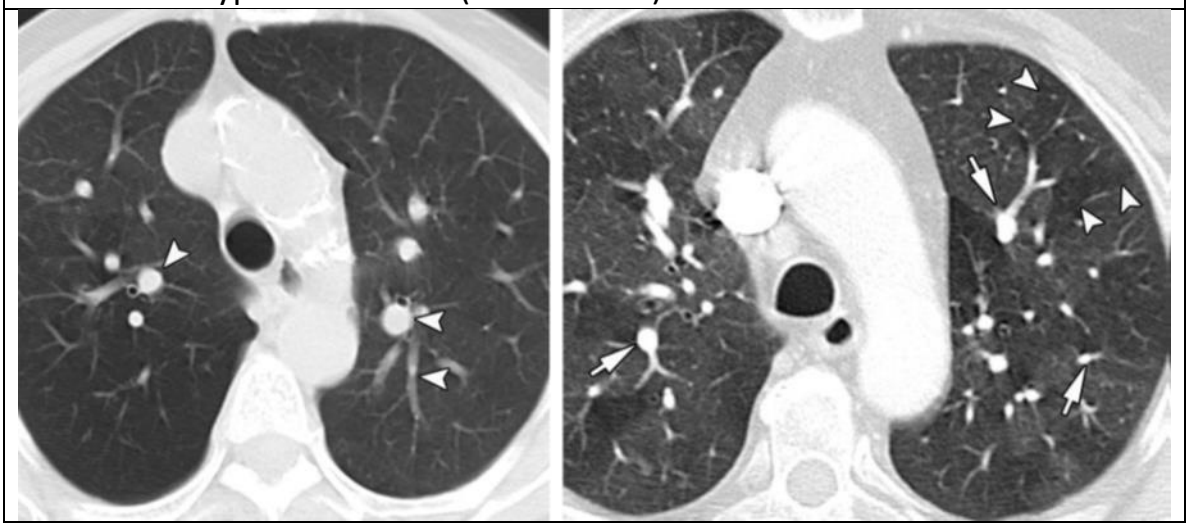
- Filling of retrosternal airspace (arrow), with right ventricular dilatation.
- The right ventricle is in contact with >1/3 of the distance from the sternodiaphragmatic angle (black arrowhead) to the point where the trachea meets the sternum (white arrowhead).



❖ Pulmonary Vascular Signs

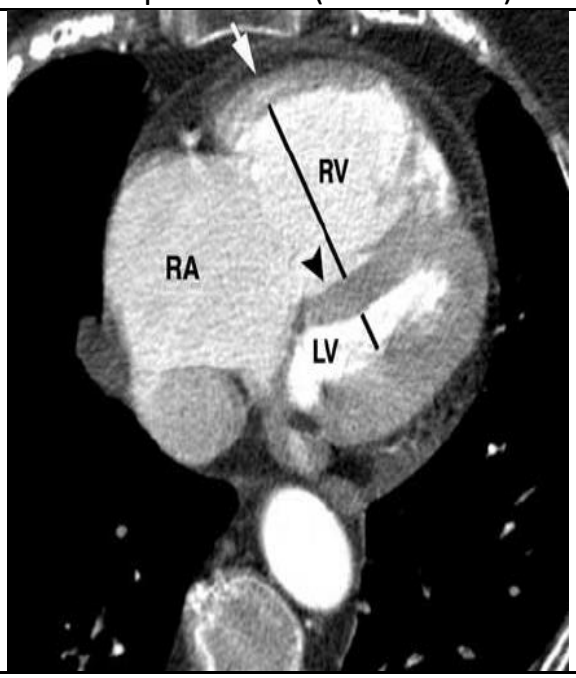
- **Dilatation of main pulmonary artery > 29 mm.**
- **PA / AA ≥ 1**
- **Pulmonary artery calcifications are pathognomonic for chronic pulmonary artery hypertension.**

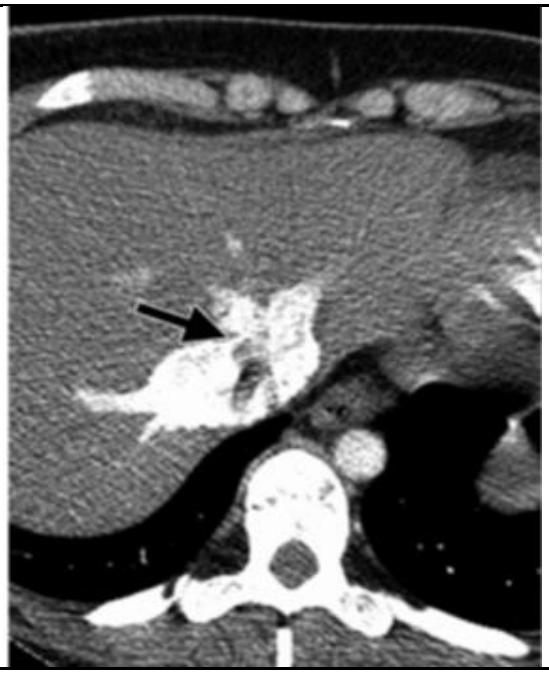
- Marked enlargement of the central pulmonary arteries (arrowheads) in relation to the bronchi.
- Peripheral prunning / tappering of pulmonary vessels
- Mosaic perfusion pattern: with increased diameters of vessels in areas of hyperattenuation (arrows) and sharp tapering of peripheral vessels in areas of hypoattenuation (arrowheads).



❖ CT Cardiac signs:

- RV hypertrophy: Myocardium thickness (white arrow) >4 mm
- RV dilatation: RV / LV diameter at the midventricular level >1
- Leftward bowing of the interventricular septum (Black arrowhead)
- Reflux of contrast material into the inferior vena cava, which is dilated, and hepatic veins (Black arrow).





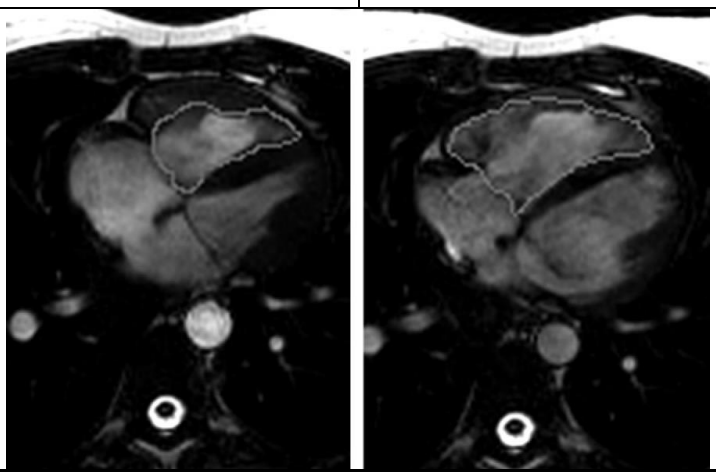
❖ Ventricular Volumes at Cine Cardiac MR Imaging

- Increased right ventricular end diastolic and end systolic volume
- Decreased right ventricular stroke volume
- Decreased right ventricular cardiac output
- Decreased right ventricular ejection fraction

- Decreased left ventricular end diastolic volume and end systolic volume
- Decreased left ventricular peak filling rate (left ventricular end diastolic volume per sec)

- Increased ventricular mass index (calculated by dividing the right ventricular mass by the left ventricular mass)

RV end-systole Volume	RV end-diastole volume



❖ Delayed MRI Contrast Enhancement (10min after contrast injection)

Typical enhancement pattern at the insertion of the right ventricle into the interventricular septum (arrows).	

Primary pulmonary hypertension (PPH) – WHO group 1, precapillary

- The pathologic hallmark of primary pulmonary hypertension (PPH) is the plexiform lesion in the wall of the muscular arteries, which is a focal disruption of the elastic lamina by an obstructing plexus of endothelial channels.
- PPH may be idiopathic (females > males) or familial
- On imaging, there is typically enlargement of the main pulmonary arteries with rapidly tapering peripheral vessels.

Pulmonary hypertension due to left-to-right cardiac shunts – WHO group 1, precapillary

- Congenital left-to-right cardiac shunts
    - Ventricular septal defect (VSD)
    - Atrial septal defect (ASD)
    - Partial anomalous pulmonary venous return

⇨ ↑ pulmonary arterial flow

⇨ Irreversible vasculopathy characterized by pulmonary hypertension

⇨ Reversal of the congenital shunt, known as Eisenmenger syndrome.

- Imaging of PAH secondary to a congenital shunt is similar to that of PPH. There is enlargement of the central and main pulmonary arteries, with peripheral tapering.

Pulmonary veno-occlusive disease – WHO group 1, postcapillary

- PAH secondary to pulmonary veno-occlusive disease is caused by fibrotic obliteration of the pulmonary veins and venules.
- Pulmonary veno-occlusive disease:
    - Idiopathic
    - Associated with pregnancy, drugs (especially bleomycin), and bone marrow transplant.
- Imaging features pulmonary arterial enlargement.
- Pulmonary edema and ground glass centrilobular nodules.

Pulmonary hypertension associated with hypoxemic lung disease – WHO group 3, precapillary

• COPD, sleep apnea, and interstitial lung disease

⇨ Chronic hypoxic vasoconstriction => vascular remodeling -> hypertrophy of pulmonary arterial vascular smooth muscle and intimal thickening.

⇨ Chronic lung disease can further contribute to obliteration of pulmonary microvasculature through emphysema and the perivascular fibrotic changes of pulmonary fibrosis.

Chronic thromboembolic pulmonary hypertension (CTEPH) – WHO group 4, precapillary

- Chronic occlusion of the pulmonary arterial bed can lead to pulmonary arterial hypertension
- Complication affecting 1–5% of patients who develop acute pulmonary embolism (PE).
- PE-protocol CT is typically the first step in workup of newly diagnosed pulmonary hypertension.

Characteristic imaging features are:

- Peripheral, eccentric filling defects in the pulmonary arterial tree.
- Fibrous strands are sometimes visible on cross-sectional imaging.
- Mosaic perfusion may be present.
- Secondary corkscrew bronchial arteries that are tortuous and dilated.

Fibrosing mediastinitis – WHO group 5, postcapillary

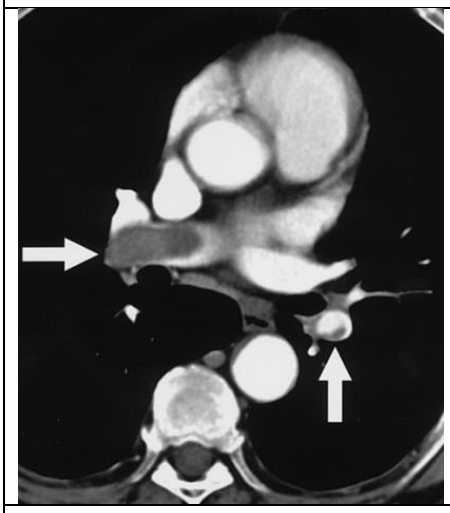
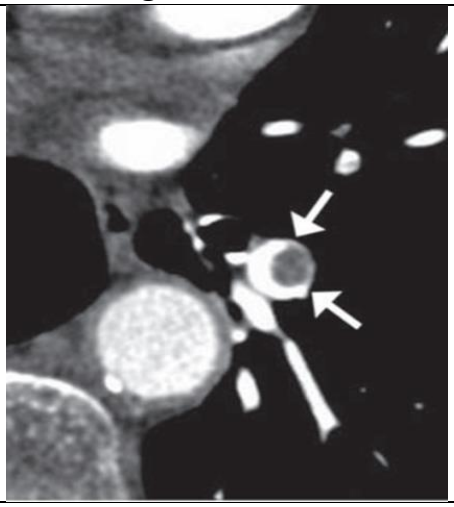
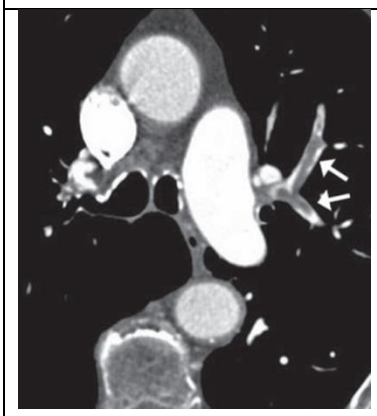
- Progressive proliferation of fibrous tissue within the mediastinum may lead to encasement and compression of mediastinal structures.
- The most common causes of fibrosing mediastinitis are histoplasmosis and tuberculosis.
- Imaging features of fibrosing mediastinitis include increased mediastinal soft tissue, often with calcified lymph nodes due to prior granulomatous infection.

Pulmonary Embolism

A) Acute PE:

❖ CT Pulmonary arteries Findings:

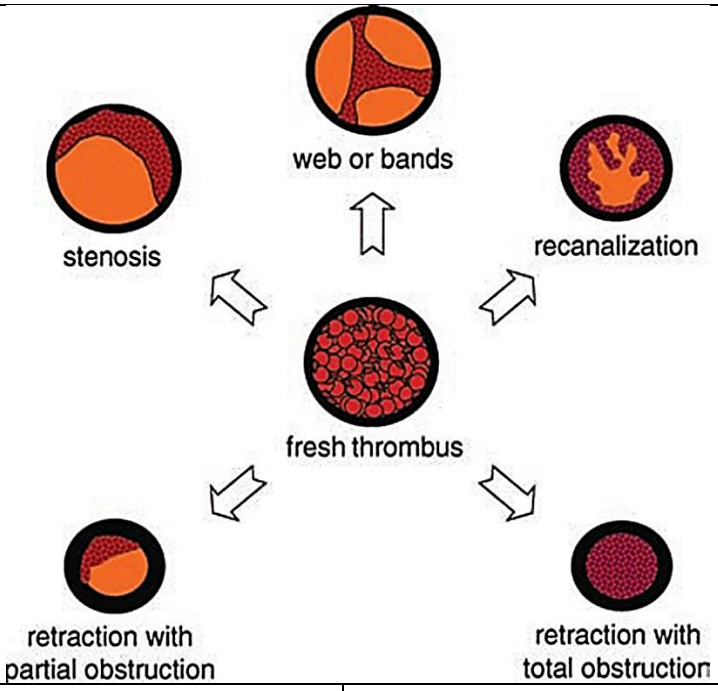
1) Complete filling defect + Enlargement of the occluded artery in comparison with adjacent patent vessels
2) Partial central filling defect surrounded by contrast material enhancement = “railway track” sign on longitudinal images
3) Partial peripheral filling defect + Acute angles with the arterial wall

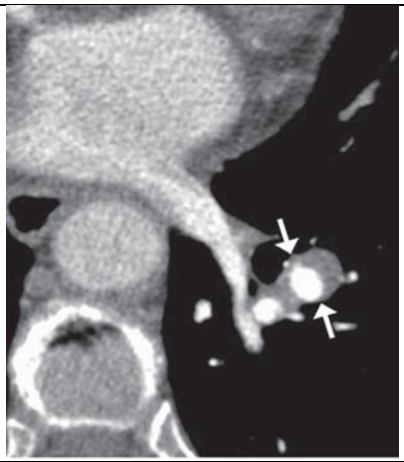
Intraluminal filling defect	Partial peripheral filling defect Acute angle with wall
	
“Railway track” sign = central partial filling defects surrounded by contrast material	Complete filling defect + Enlargement of vessel
	

B) Chronic Pulmonary Thromboembolism

- is mainly a consequence of incomplete resolution of pulmonary thromboembolism.
- Increased vascular resistance due to obstruction of the vascular bed leads to pulmonary hypertension and cor pulmonale.

❖ CT Pulmonary artery signs:



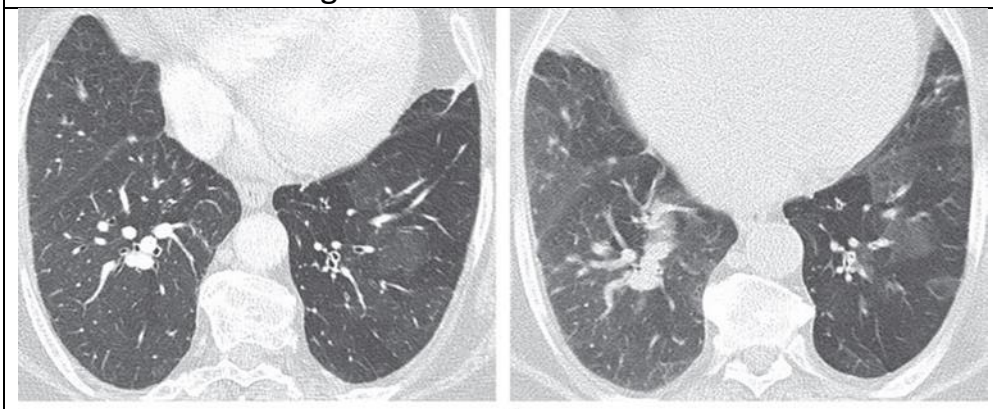
Complete occlusion and marked reduction in size	Partial obstruction with eccentric thrombus forming obtuse angle with the vessel wall
	

❖ CXR signs:

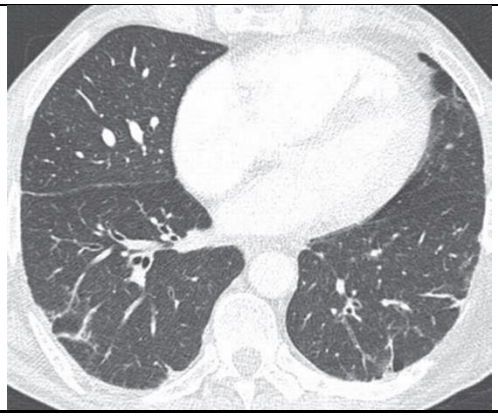
- Westermark sign: Decreased vascularity in the peripheral lung
- Fleischner sign: Enlargement of the central pulmonary artery
- Hampton sign: pleura-based areas of increased opacity
- Hemidiaphragm elevation
- Focal areas of increased opacity,
- Linear atelectasis, and
- Pleural effusion.

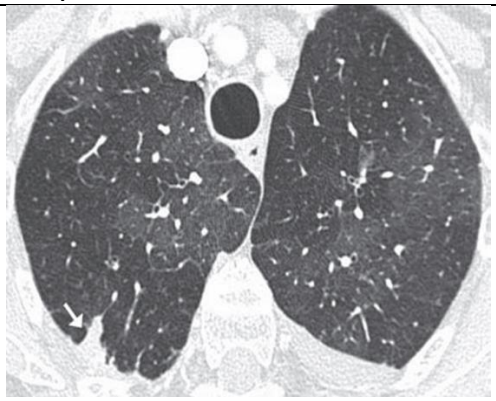
❖ CT Chest parenchymal signs:

- Wedge shaped peripheral consolidations
- Mosaic attenuation pattern:
    - Decreased vessel size in areas of low attenuation compared to areas of high attenuation.



Peripheral lineal atelectasis	Peripheral subpleural wedge-shaped area of consolidation





❖ CT signs of Pulmonary Hypertension (See Pulmonary Hypertension)

Paradoxical Embolism

Triad of:

1) Systemic embolism
2) Venous thrombosis,
3) Intra-cardiac or intra-pulmonary abnormal communication between the right and left circulations

Intracardiac
PFO
Iatrogenic connection (baffle defect, Fontan conduit, Rashkind device)
Enlarged thebesian veins (ie, interatrial muscle bundle)
Congenital anomaly (atrial septal defect, unroofed coronary sinus, ventricular septal defect, atrioventricular septal defect)
Extracardiac
Pulmonary arteriovenous malformation (congenital, secondary to cavopulmonary shunts)
Systemic to pulmonary venous communication (congenital, acquired)
Arterioarterial communication (patent ductus arteriosus) or venovenous communication

PFO = Patent Foramen Ovale

Pulmonary Artery

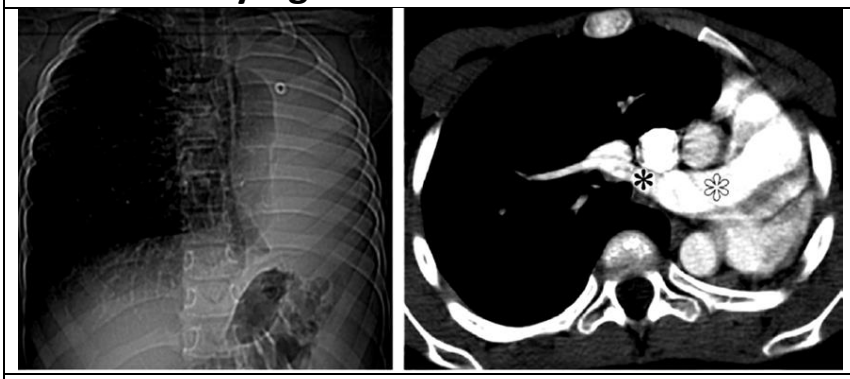
Pulmonary Artery Congenital Anomalies

1) Pulmonary Underdevelopment

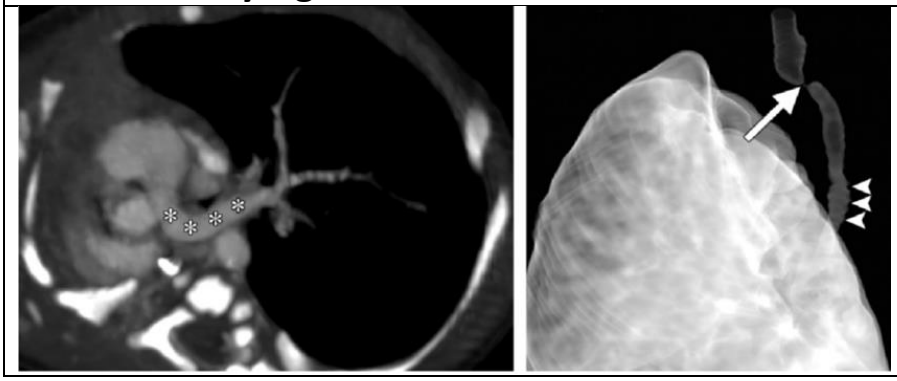
Classification of Pulmonary Underdevelopment

Category	Description
Pulmonary agenesis	Complete absence of the lung parenchyma, bronchus, and pulmonary vasculature
Pulmonary aplasia	Blind-ending rudimentary bronchus is present, without lung parenchyma or pulmonary vasculature
Pulmonary hypoplasia	Bronchus and rudimentary lung are present; however, the airways, alveoli, and pulmonary vessels are decreased in size and number

Lt Pulmonary Agenesis



Rt Pulmonary Agenesis



2) Proximal Interruption of the Pulmonary Artery

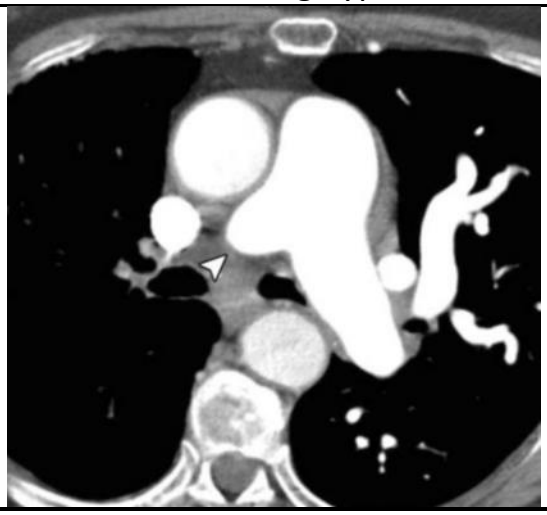
- The proximal portion of the main pulmonary artery (arising from the primitive sixth aortic arch) fails to appear during embryologic development.
- RT > LT

Compensatory systemic vessels:

- Bronchial arteries
- Transpleural branches of the intercostal, internal mammary, subclavian, and innominate arteries

Imaging Findings:

- The mediastinal portion of the pulmonary artery may be completely absent or may terminate within 1 cm of its origin.
- Ipsilateral lung volume loss
- Ipsilateral mediastinal shift
- Contralateral lung hyperinflation.



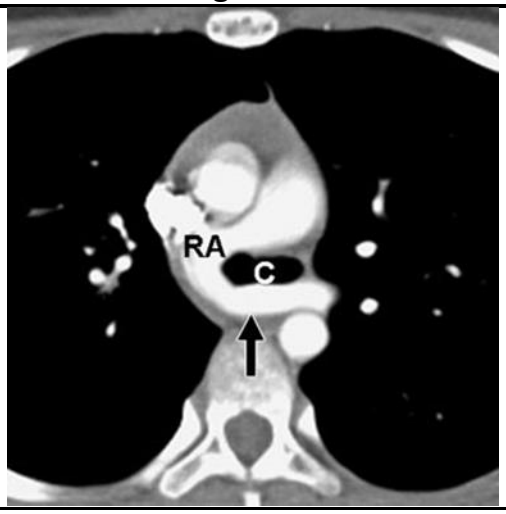
3) Pulmonary Artery Sling

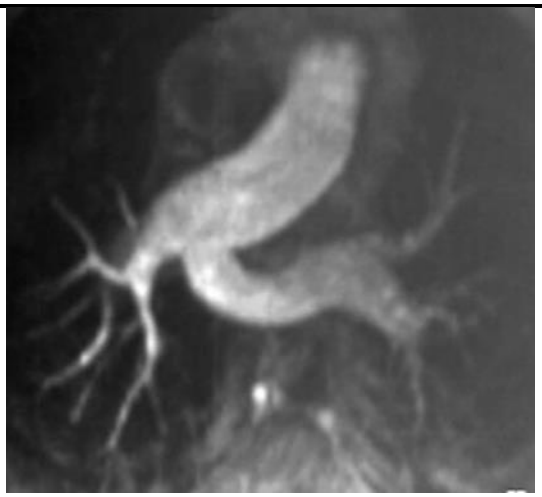
The left main pulmonary artery

⇨ Arises from the proximal right main pulmonary artery

⇨ Courses between the trachea and esophagus

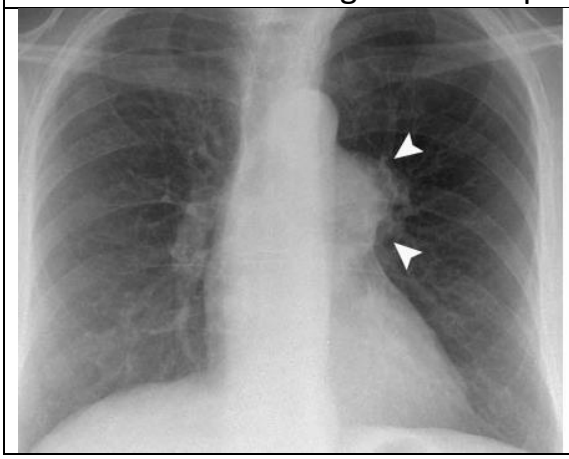
⇨ Forms “Sling” around distal trachea.

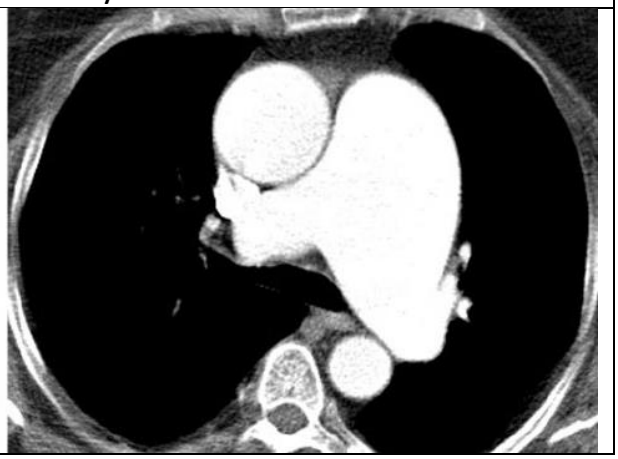




4) Idiopathic Dilatation of the Pulmonary Trunk

- Rare congenital anomaly
- Abnormal enlargement of the pulmonary trunk, with or without dilatation of the right and left pulmonary arteries





5) Patent Ductus Arteriosus (PDA)

- Abnormal persistence of the normal in utero fetal pulmonary-systemic connection.

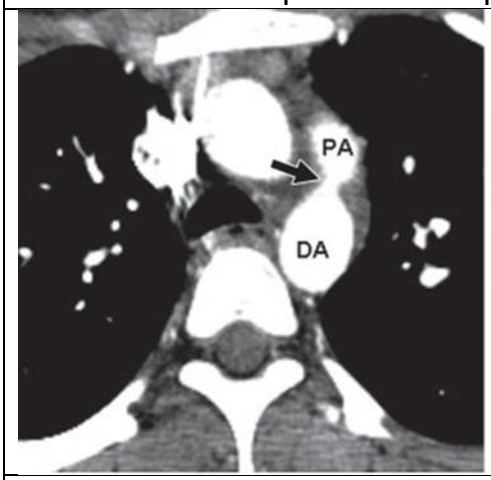
- Acyanotic left-to-right shunt between the aorta and pulmonary artery.

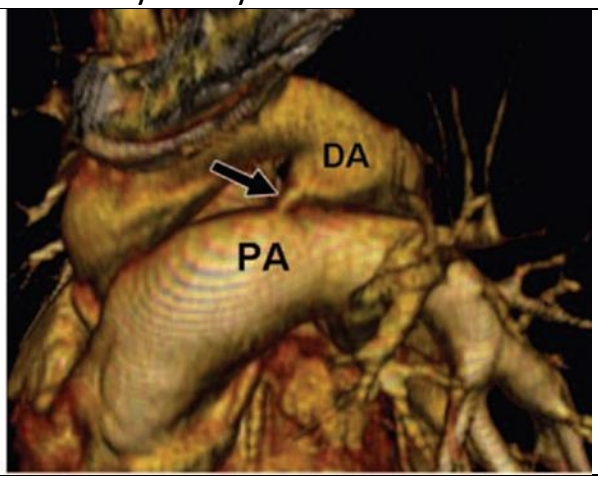
- PDA closes functionally 18–24 hours after birth.
- The anatomic closure occurs at 1 month of age.

- When a PDA closes, it forms the ligamentum arteriosum, which may calcify.
- Linear calcification in the region of aortopulmonary window is a calcified ligamentum arteriosum

Imaging findings:

- Tubular structure connecting the descending proximal aorta with the distal main or proximal left pulmonary artery.





6) Truncus Arteriosus

Pulmonary AVM

- Direct, abnormal communication between the pulmonary artery and pulmonary vein.
- Most common congenital

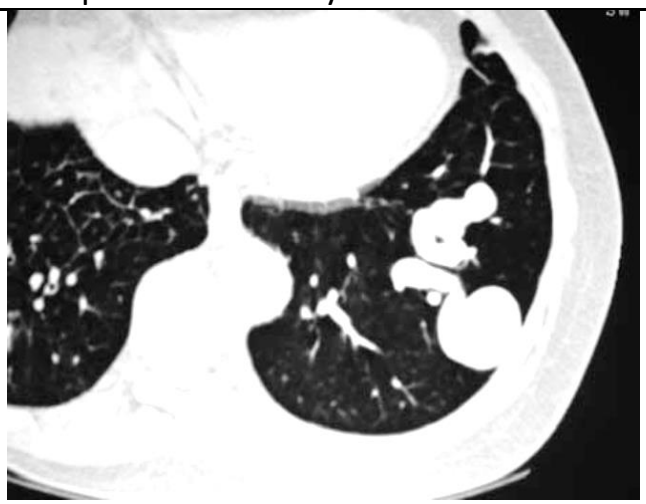
- ➢ 2/3 of PAVM has a lower lobe predilection
- ➢ 2/3 of PAVM are multiple lesions
- ➢ 35% are associated with Hereditary hemorrhagic telangiectasis (HHT) = Rendu-Osler-Weber Syndrome

Classification of PAVM:

a) **Simple type of PAVM**: Single segmental artery and draining vein.
b) **Complex type of PAVM:** ≥ 2 supplying arteries and draining veins.

Treatment options:

- ✓ Embolization: using coils.
- ✓ Surgical: Vascular ligations, local resection, segmentectomy, lobectomy, or pneumonectomy.



Pulmonary Artery Aneurysm

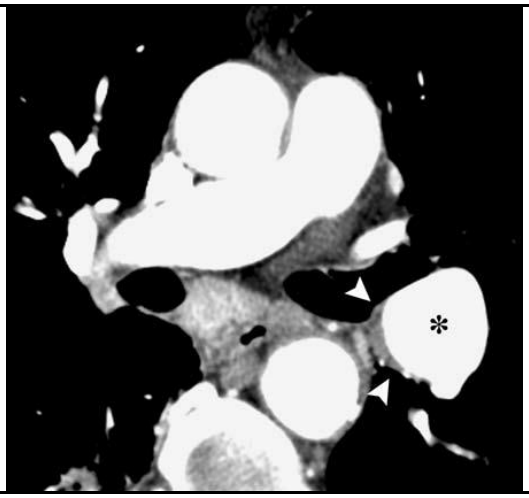
- Congenital
- Infection (mycotic aneurysm)
- Trauma
- Vascular abnormality (eg, cystic medial necrosis, Behçet disease, Marfan syndrome, and Takayasu disease)
- Pulmonary hypertension

❖ Behçet Disease

Chronic inflammatory disorder of systemic vasculitis of unknown origin.
Characterized by:

- Recurrent oral and genital ulcerations
- Ocular anomalies: Uveitis
- Systemic vasculitis

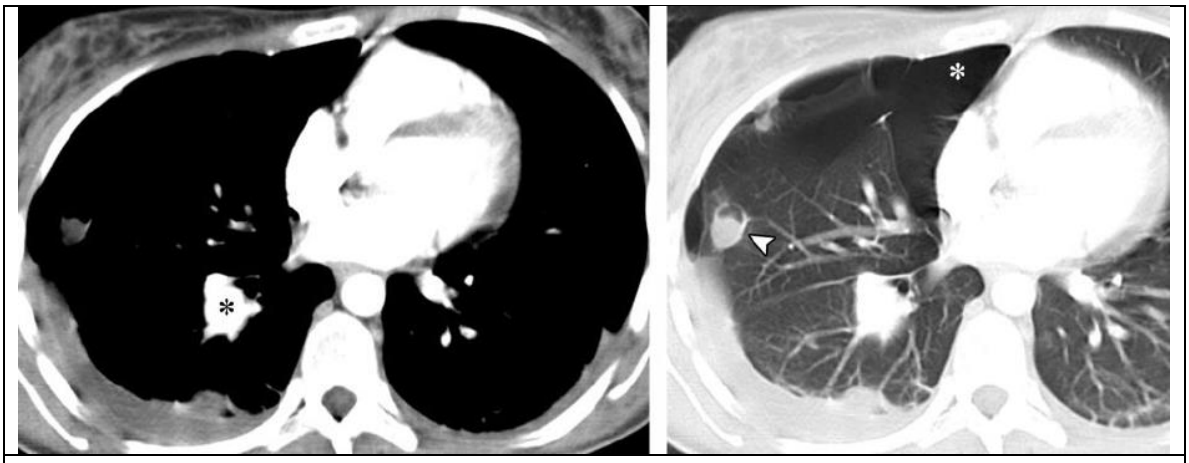
- Single or multiple
- Central pulmonary artery aneurysms
- Mural thrombosis



❖ Mycotic Aneurysm

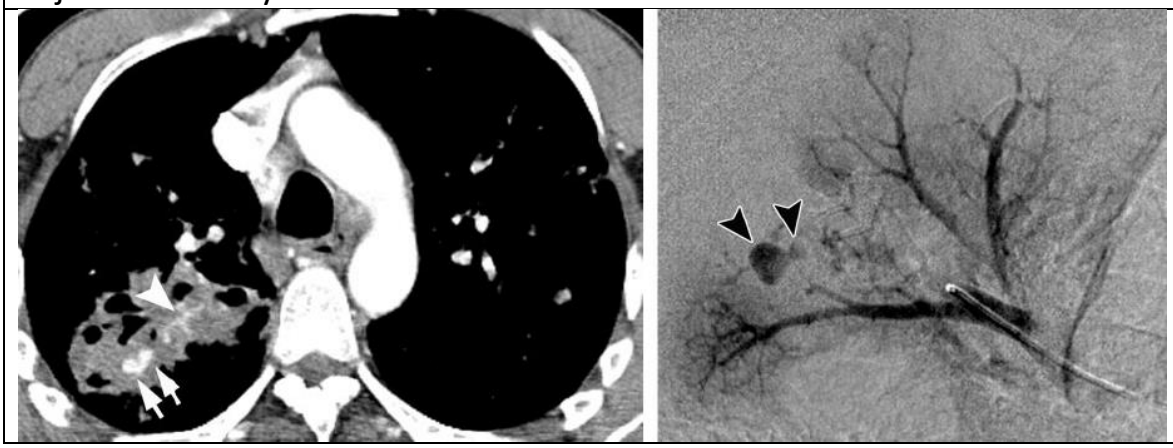
Secondary to:

- Endovascular seeding (due to septic pulmonary emboli) seen in patients with endocarditis.
- Direct extension of infection from the adjacent parenchyma are seen in patients with necrotizing pneumonia or chronic tuberculosis



❖ Rasmussen Aneurysm

A rare condition caused by weakening of the pulmonary artery wall from adjacent cavitary tuberculosis



Takayasu Arteritis

Idiopathic arteritis that mainly affects the elastic arteries.
young Asian women
Location:

- ✓ **Aorta and its major branches**
- ✓ **Pulmonary arteries.**

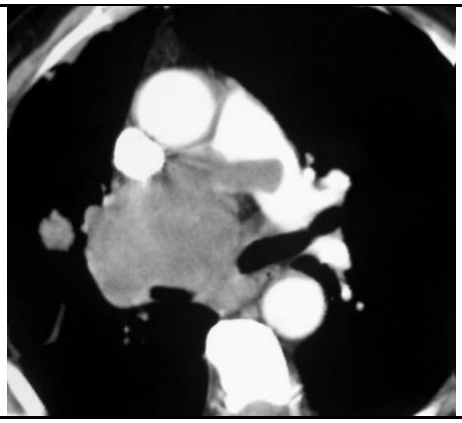
Active phase: Wall thickening + enhancement
Chronic phase: Mural calcification + Luminal stenosis or occlusion

PA Primary Sarcoma

Type: Undifferentiated sarcoma and leiomyosarcoma.
Location: Main or proximal pulmonary arteries

Imaging Findings:

Intraluminal filling defect + Enlargement of the vessel
± Extravascular invasion



DD: PA Dilatation

1) Idiopathic pulmonary artery dilatation.
2) Pulmonary stenosis.
- **Poststenotic dialatation of the main PA + Lt PA**

3) Pulmonary Aneurysm: Behçet
- **Behçet triad: Genital & oral ulcers + Uveitis + Systemic vasculitis.**

4) Pulmonary Arterial Hypertension.

DD: PA Stenosis

1) **Pulmonary carcinoma**
2) **Mediastinal fibrosis**
3) **Takayasu arteritis**

Pulmonary Vein

A. Congenital Disease:

(a) Anomalies in the number or diameter of the vessels
(b) Abnormal drainage or connection with the pulmonary arterial tree.

B. Acquired Disease:

(a) Stenosis and obstruction
(b) Hypertension
(c) Thrombosis
(d) Calcifications
(e) Collateral circulation

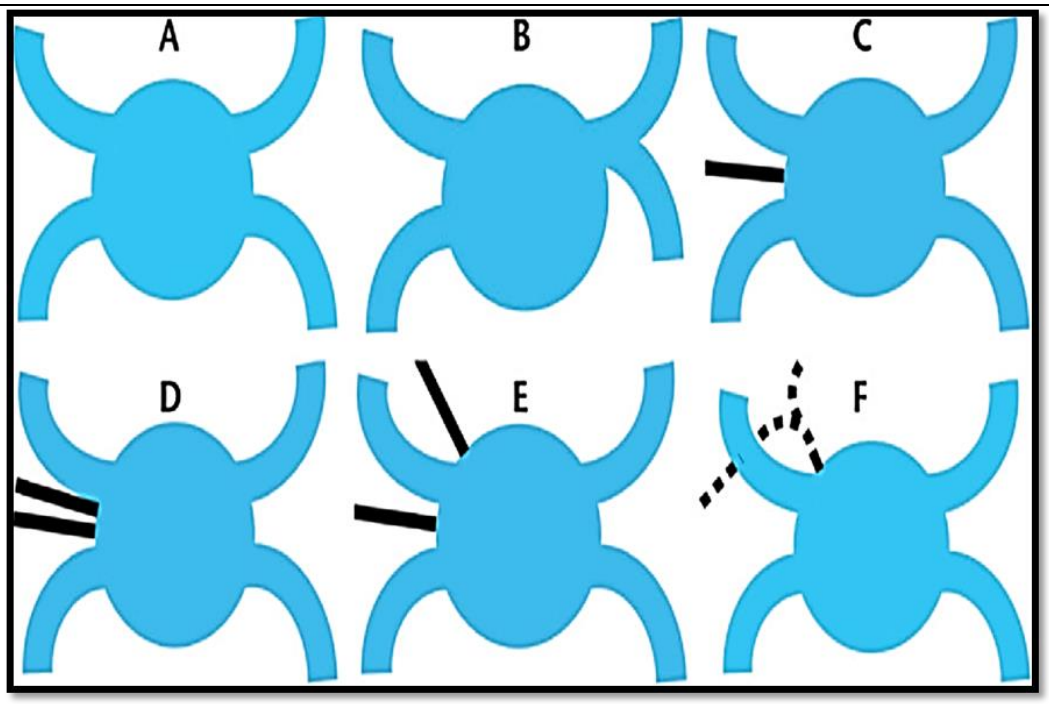
C. Atrial Fibrillation:

The pulmonary veins contribute to originating and maintaining atrial fibrillation.

D. Pulmonary Neoplasm:

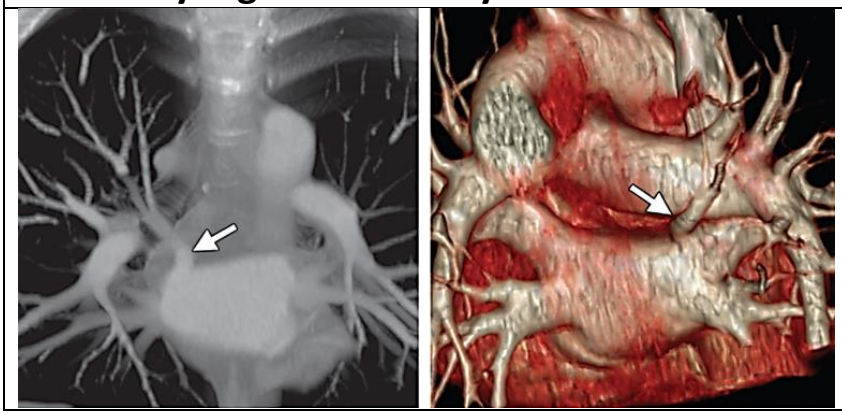
Non–small cell lung cancer, extension into the intrapericardial portion of the pulmonary veins => T4 staging.

Normal Pattern & Variations Of Pulmonary Vein Anatomy



Solid black lines are variant accessory veins.
A) Typical normal pattern.
B) Left common trunk.
C) 1 Accessory right middle pulmonary vein.
D) 2 Accessory right middle pulmonary veins.
E) 1 Accessory right middle pulmonary vein and 1 accessory right upper pulmonary vein.
F) Right top pulmonary vein (dashed black line).

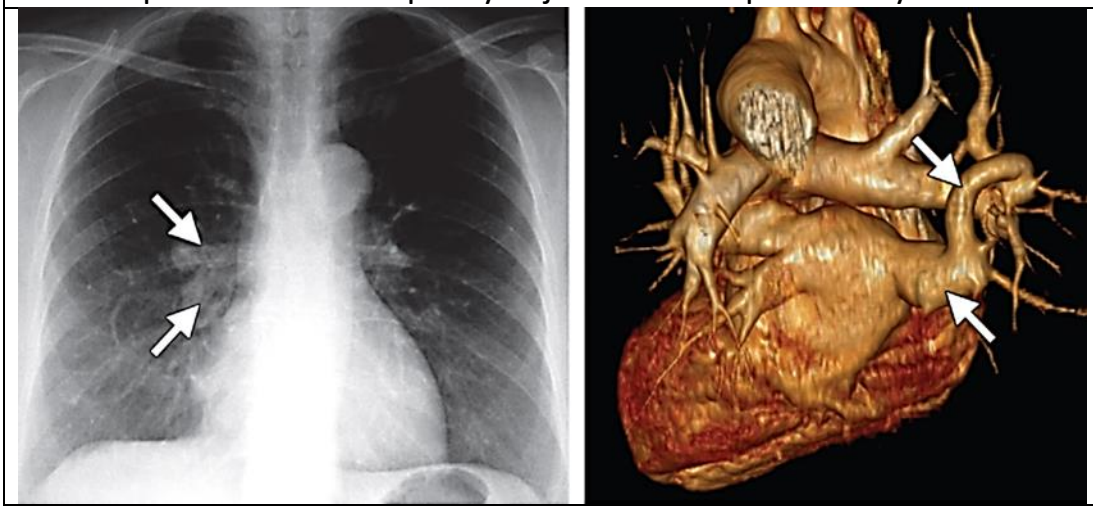
Accessory Right Pulmonary Vein



❖ Anomalous Unilateral Single Pulmonary Vein

Abnormal solitary pulmonary vein that joins one side of the left atrium after uniting the pulmonary veins from one lung

Rt > Lt > Bi
Juxtahilar location
CXR: serpentine tubular opacity adjacent to the pulmonary hilum

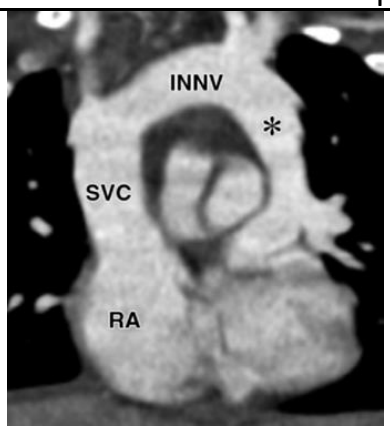


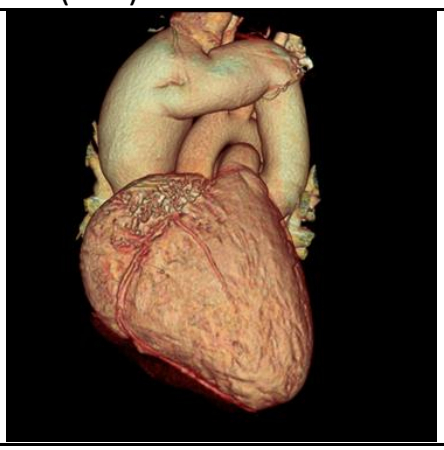
❖ Total Anomalous Pulmonary Venous Return (TAPVR)

Result from failure of the common pulmonary vein (CPV) to connect to the left atrium, with persistence of the primitive splanchnic connections of the pulmonary veins to the cardinal systemic veins => Right atrium

A) Supracardiac TAPVR: (50%)

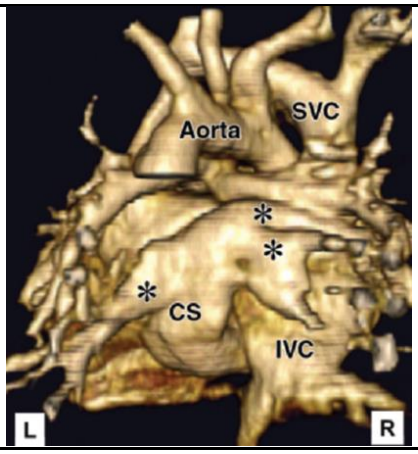
Pulmonary veins => pulmonary vein confluence => Ascending vertical vein => Innominate vein => Superior vena cava (SVC) => Rt Atrium.





B) Cardiac TAPVR:

Pulmonary venous confluence connects directly to => Right atrium or coronary sinus (CS).



C) Infracardiac TAPVR

The pulmonary venous confluence drains to systemic veins below the diaphragm

- ⇨ Portal vein (at the confluence of the splenic and superior mesenteric veins).
- ⇨ Ductus venosus
- ⇨ Hepatic veins
- ⇨ Inferior vena cava (IVC).

❖ Partial Anomalous Pulmonary Venous Return (PAPVR)

Some but not all segments of a developing lung fail to establish connections with the common pulmonary vein (CPV) and instead retain their connections to the primitive splanchnic system of the cardinal veins.

PAPVR drain into:

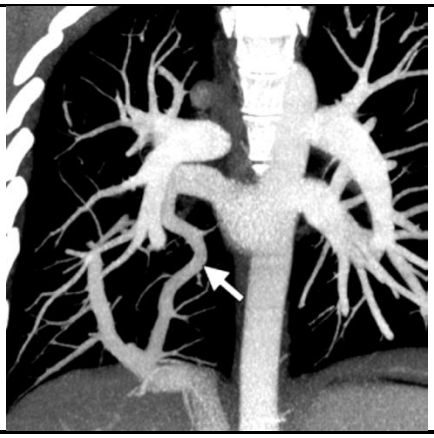
- → Infradiaphragmatic IVC
- → Suprahepatic portion of IVC.
- → Hepatic vein
- → Portal vein
- → Coronary sinus
- → Right atrium

Left upper lobe pulmonary vein drain into => left brachiocephalic vein.	Descending anomalous vein (arrow) draining below the diaphragm. ⇨ IVC ⇨ Hepatic veins.

❖ Levoatriocardinal Vein

Anomalous connection between the left atrium and a vein from the systemic venous system

Anomalous vein (arrow) connecting the left atrium to IVC



❖ Sinus Venosus Defect

Sinus venosus defect is considered to represent unroofing of the right pulmonary veins into the SVC or IVC.

SVC type:

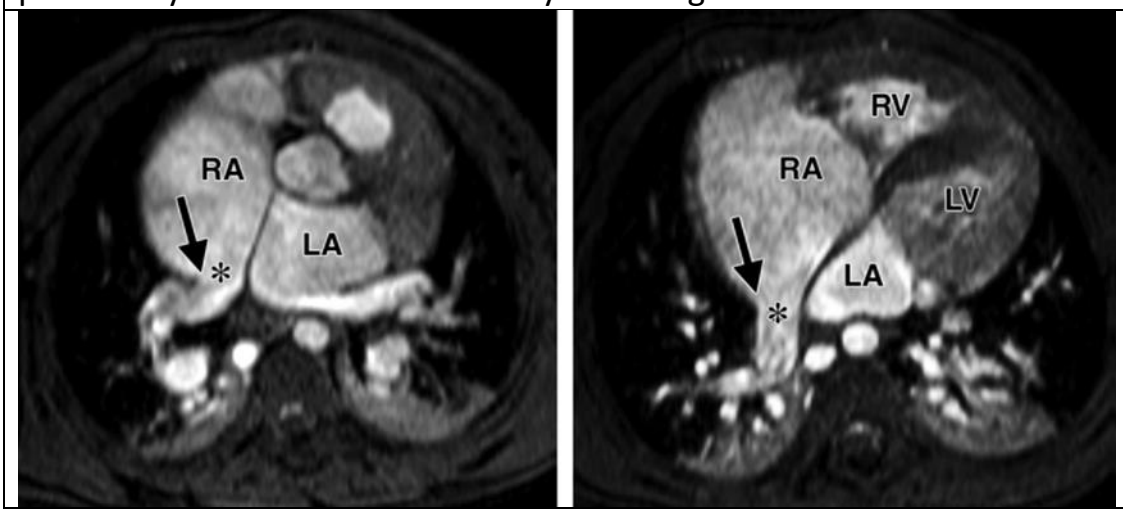
- Unroofing of the right upper pulmonary vein (RUPV) into the SVC

IVC type

- Unroofing of the right lower pulmonary vein into the IVC

❖ Malposition of Septum Primum

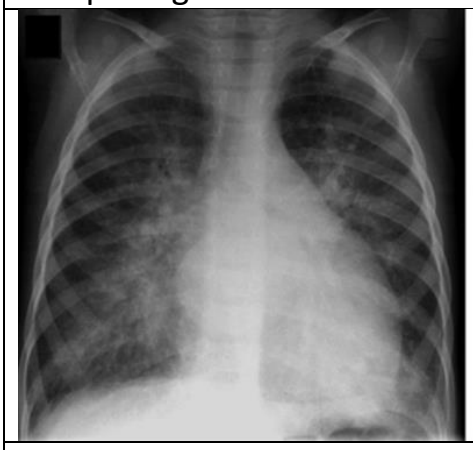
The septum primum deviated leftward => Normally connected right pulmonary veins drain anomalously to the right atrium

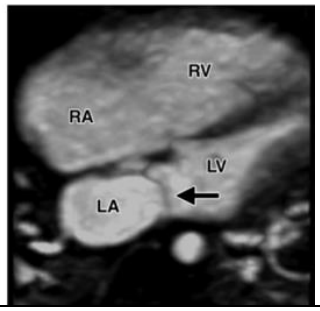


❖Cor Triatriatum

Result from defective resorption of the CPV into the left atrium, with associated stenosis of the CPV orifice

- ⇨ Accessory chamber within the left atrium that represents the CPV.
- ⇨ This accessory chamber communicates with the left atrium through an opening that demonstrates varying degrees of stenosis



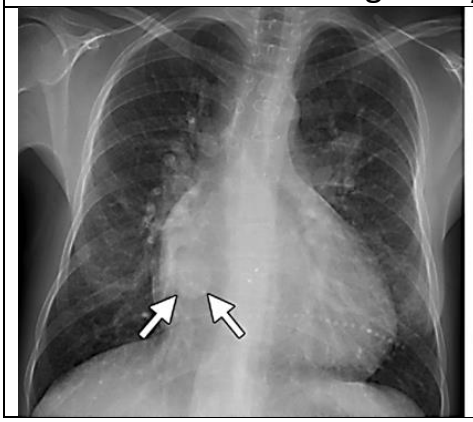


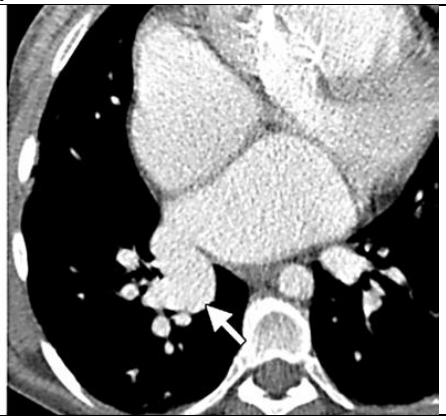
❖ Pulmonary Varix

Pulmonary varix is an enlarged pulmonary vein without a large feeding artery or nidus.
Typically observed near its point of entry into left atrium.

Pulmonary varices may be congenital or acquired.
Acquired pulmonary varices are associated with chronic pulmonary hypertension and disease of the mitral valve

- Pulmonary varix can mimic a solitary pulmonary nodule
- Contiguity of pulmonary varix with the adjacent pulmonary vein and concurrent contrast material filling within these structures
- Absence of a feeding artery or nidus





❖ Pulmonary Vein Stenosis

Congenital Pulmonary Vein Stenosis:

is a condition in which there is uncontrolled growth of myofibroblast- like connective tissue cells

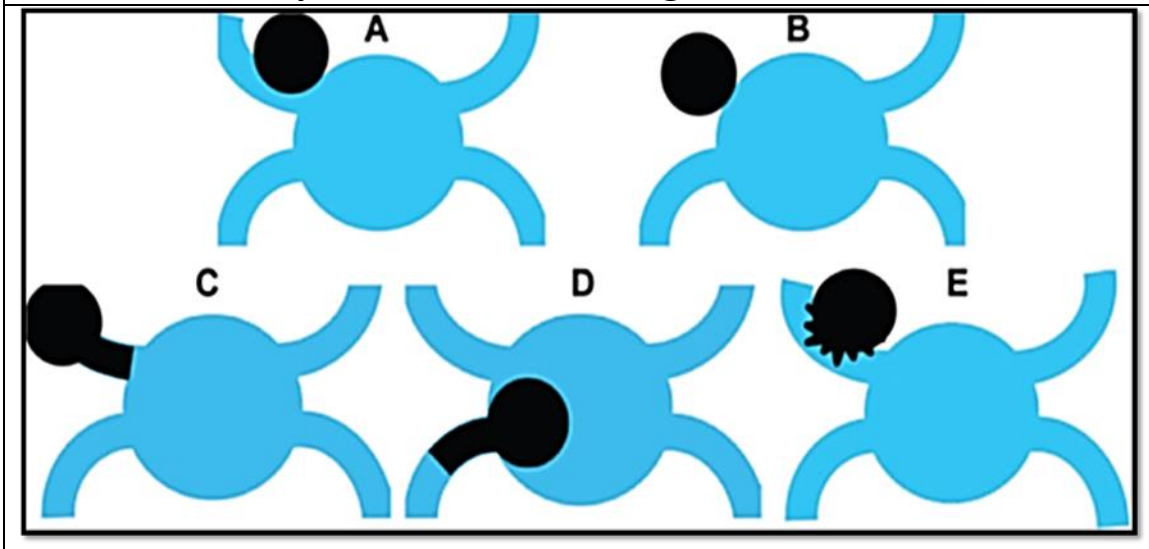
⇨ Pulmonary vein wall thickening + lumen narrowing

Acquired Pulmonary Vein Stenosis:

Most common causes:

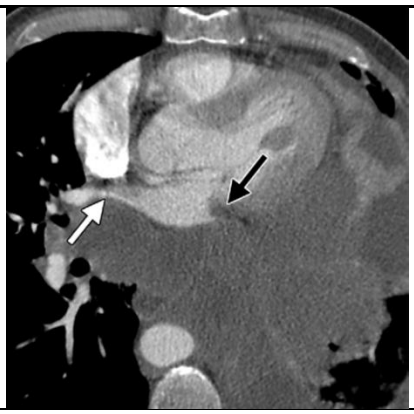
- RFA Complications
- Neoplastic Infiltration
- Fibrosing Mediastinitis
- Infiltration or extrinsic compression by a benign inflammatory process (eg, sarcoidosis or tuberculosis

Patterns of Neoplasm-related Findings

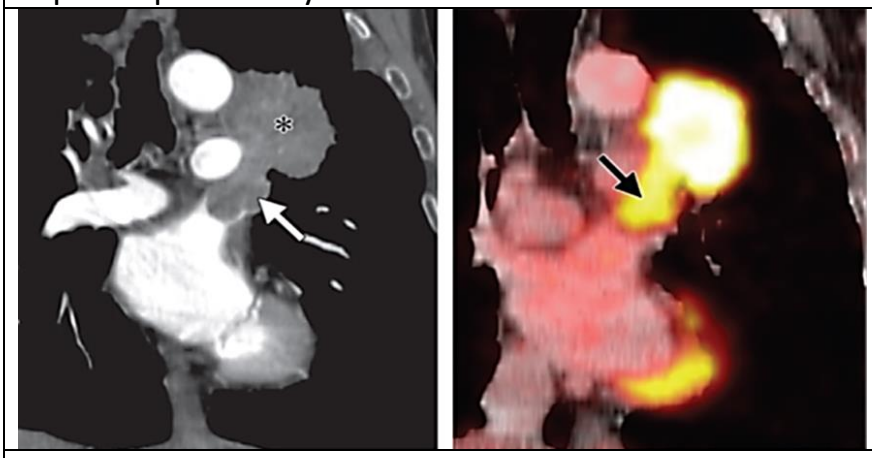


A) Extrinsic compression.
B) Complete obstruction.
C) Extracardiac neoplasm with tumor thrombus.
D) Cardiac neoplasm with tumor thrombus.
E) Neoplastic infiltration of the pulmonary vein wall without complete obstruction

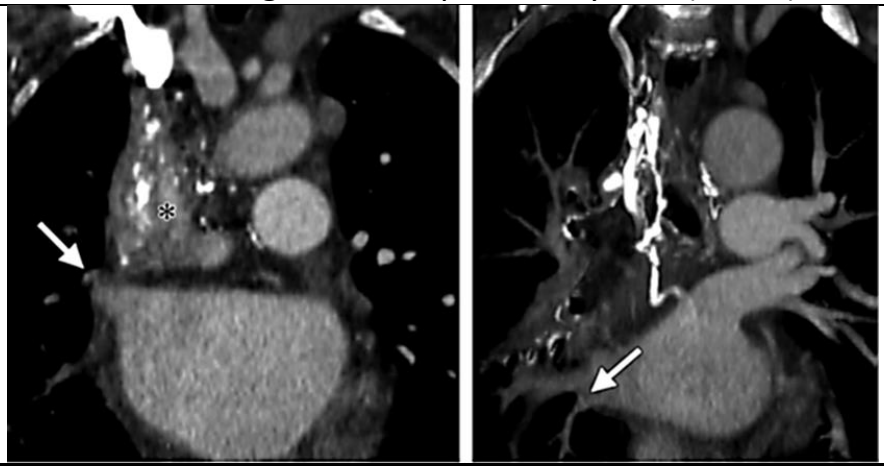
Mediastinal mass causing
Extrinsic compression of right inferior pulmonary vein (white arrow)
Complete obstruction of left inferior pulmonary vein (black arrow)



Non–small cell lung neoplasm (*) => Tumor thrombus (arrow) in left superior pulmonary vein



Fibrosing Mediastinitis
Right paratracheal mass (*) with calcifications
Obstructs SVC => SVC Syndrome
Stenosis of the right superior pulmonary vein (arrow).
Stenosis of the right inferior pulmonary vein (arrow).



Pleura Anatomy

Parietal pleura:

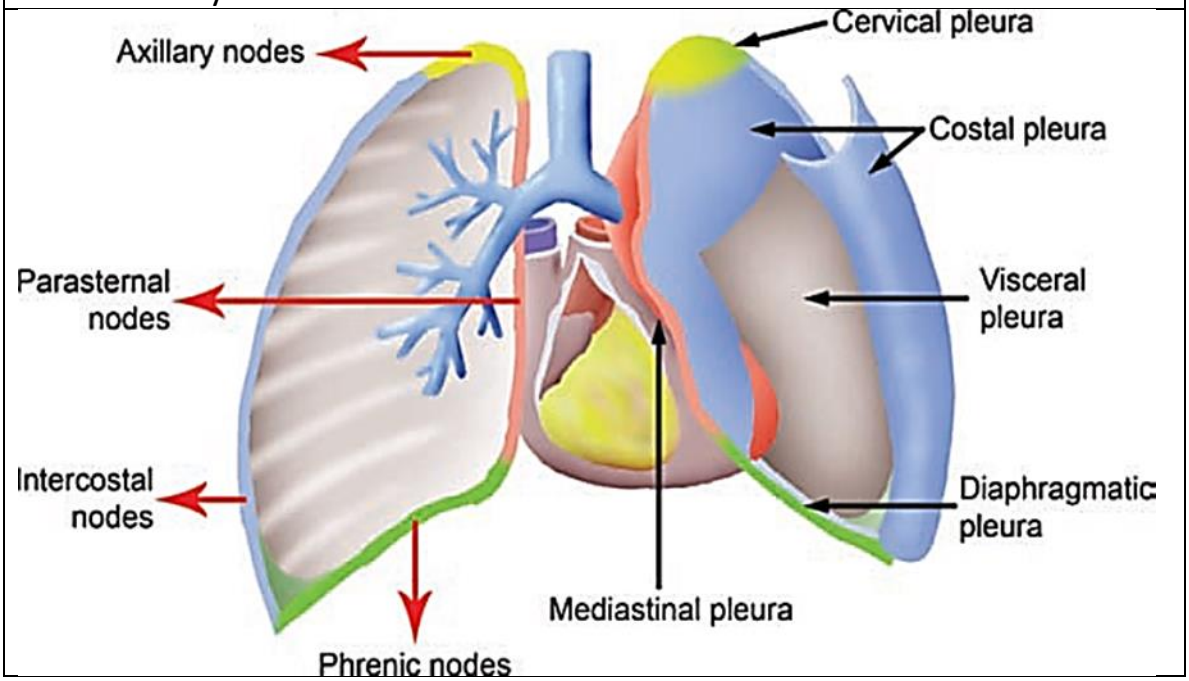
- Thoracic wall (costal pleura),
- Lateral aspect of the mediastinum (mediastinal pleura)
- Thoracic inlet (cervical pleura),
- Thoracic surface of the diaphragm (diaphragmatic pleura).

Visceral pleura:

- Covers the lungs
- Form the interlobar, minor, and accessory fissures

Lymphatic drainage:

- Thoracic wall (intercostal, parasternal, mediastinal, and phrenic LNs)
- Axillary LNs



Pleural Effusion

Types & Causes:

1) Transudate

Caused by systemic or local imbalances in hydrostatic and oncotic forces.
Common causes include:

- Hypoproteinaemia (Nephrotic syndrome – Hepatic C – Anaemia)
- Heart failure

2) Exudate

- No reliable imaging features to distinguish between transudative and exudative effusions.
- An exudative effusion is distinguished from a transudate by thoracentesis.
- Analysis of the thoracentesis fluid by Light's criteria is the most reliable method to distinguish transudate from exudate.
- If any one of the following is present, the effusion is classified as an exudate:
  - Ratio of pleural fluid protein to serum protein >0.5.
  - Ratio of pleural fluid LDH to serum LDH >0.6.
  - Pleural fluid LDH >2/3 upper limits of normal for serum.
- Causes:
  - Pneumonia with parapneumonic effusion, empyema,
  - Tuberculous pleuritis.
  - Mesothelioma or pleural metastasis.
  - Rheumatoid arthritis or other collagen vascular diseases.
  - Pancreatitis

3) Haemothorax:

- Caused by:
  - Franck blood in pleural sac (Trauma – Post operative – Blood disease)
  - Blood stained effusion (Pulm. infarction – Bronchial carcinoma)

4) Chylothorax

- A chylothorax is a pleural effusion consisting of intestinal lymph
- Most commonly caused by obstruction of the thoracic duct:
  - Filariasis – Trauma – Malignant invasion
- Chylothorax is also associated with lymphangioleiomyomatosis (LAM).

Radiological features:

A) Free Pleural Effusion:

Small effusion:

- < 100cc: Detected by lateral decubitus view, US, CT
- 100-2000cc: Detected in lateral CXR => posterior costophrenic recess
- >200cc: Detected in PA CXR => Blunting of lateral costophrenic angle

Moderate effusion:

- Homogenous opacity + No lung markings
- Obliterating costophrenic recess
- Well defined concave upper edge
- Rising laterally > medially (towards axilla)

Massive effusion:

- Complete or partial opacification of the hemithorax
- Collapse of the lung towards hilum
- Contralateral mediastinal shift

US:

- Anechoic

CT:

- Fluid attenuation (less than pleural thickening)

Lamellar effusion:

- Common in children and patients with heart disease
- Shallow collection between visceral pleura and lung surface
- Sometimes spares costophrenic recess.

Sub-pulmonic effusion:

- Effusion accumulate between diaphragm and undersurface of the lung
- False impression of elevated hemi-diaphragm
- Change in posture in decubitus and supine films: the fluid move in free pleural space
- On LT side => increased distance between gastric air bubble and lung base >2cm
- On RT side => large effusion may collect in the azygo-esophageal recess and minimc retrocardiac mass

B) Loculated or Encysted Pleural Effusion:

Costal encysted pleural effusion:

- Profile view: well defined biconvex opacity with peripheral border adjacent to the chest wall.
- En-face view: radio-opacity of relatively low density

Encysted Horizontal fissural pleural effusion:

- Lenticular, oval or rounded shadow with well defined edge

Encysted Oblique fissural pleural effusion:

- PA: rounded or oval shadow with well defined lower border and ill defined upper margin
- Lateral: lenticular well defined shadow along fissure

DD: Pleural effusion & Ascites by CT

1) Displaced crus sign:

- **P**leural fluid:
    - **P**osterior to diaphragmatic crus
    - Displaces crus anteriorly
- **A**scites:
    - **A**nterior to the crus
    - Displaces crus posteriorly

2) Diaphragm Sign:

- Pleural fluid => on the outer surface of the dome of the diaphragm
- Ascites => within dome of the diaphragm

3) Interface sign:

- Interface between liver or spleen with ascites is more sharp than interface between liver or spleen with pleural fluid.

4) Bare area sign:

- Ascites: peritoneal coronary ligament prevents ascites from extending over entire posterior surface of the liver.
- Pleural fluid: extends over entire posterior costophrenic recess behind liver.

❖ Empyema:

- Infected exudative pleural effusion containing pus

Causes:

- Lung: Para-pneumonic (Post infection)
- Chest wall: Osteomyelitis
- Below diaphragm: Subdiaphragmatic abscess
- Mediastinum: Broncho-pleural fistula

CT findings:

- Higher attenuation value of pleural fluid collection.
- **Split pleural sign:**
  - Enhancement & thickening of parietal and visceral pleura
  - Loculated lentiform pleural fluid collection.
- **Gas in empyema:**
  - Broncho-pleural fistula
  - Gas forming organisms

Complications:

- Broncho-pleural fistula.
- Empyema necessitates.

Pleural Thickening

Causes:

- Pleurisy: primary or secondary to lung infection or infarction.
- Previous TB infection
- End result of empyema and haemothorax.
- Post-radiotherapy of chest
- Asbestosis
- Old age
- Malignancy

Pleural Calcification

Causes:

- TB
- Asbestosis
- Post-empyema
- Post-haemothoax
- Talc

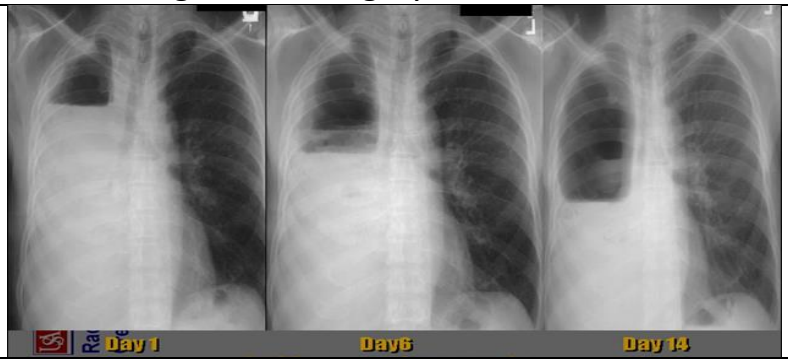
Bronchopleural Fistula (BPF)

Causes:

- Complication of pneumonectomy
- Rupture of lung abscess, empyema, TB
- Carcinoma of nearby bronchus
- Penetrating chest trauma

Radiological features:

- Picture similar to hydropneumothorax
- If persist or enlarges after surgery => BPF is considered



Pneumothorax

Air within pleural cavity due to defect in parietal or visceral pleura

❖ Causes & Types:

A) Spontaneous Pneumothorax:

Is the commonest type

Children => Staph pneumonia
Young adults => Congenital pleural bleb
Old age => Emphysema or chronic bronchitis
Lung abscess: TB – fungal – septic emboli
Cavitating neoplasm

B) Traumatic Pneumothorax:

- **Chest trauma:**
  - Blunt trauma: Road Traffic Accident (RTA) – Rib #
  - Penetrating trauma: Bullet – Stab wound
- **Iatrogenic:**
  - Pleural or lung biopsy
  - Bronchoscopy & esophagoscopy
  - CVP catheter introduction

❖ Radiological Appearance:

Small Pneumothorax:

- Small pneumothorax collects at the apex
- Radiolucent devoid of lung markings
- White line = visceral pleural line

Large Pneumothorax:

- Lung retraction
- Mediastinal shift to the contralateral side with expiration
- Deep lateral costophrenic sulcus

Tension Pneumothorax:

- Hyperexpanded hemithorax
- Lung squashed against mediastinum
- Mediastinal shift to the contralateral side with inspiration
- Depressed ipsilateral diaphragm
- Deep lateral costophrenic sulcus

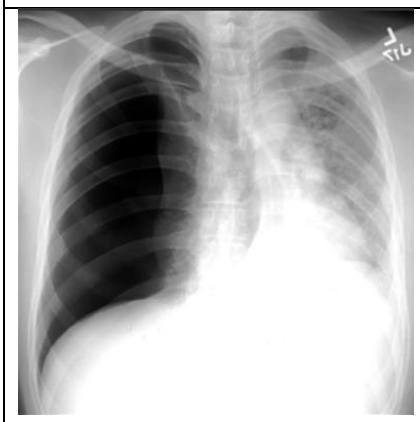
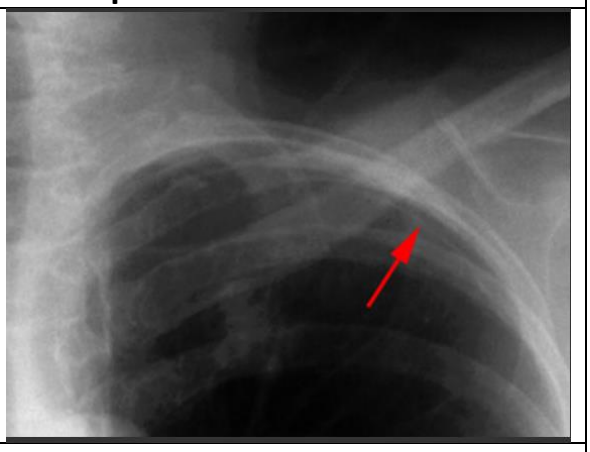
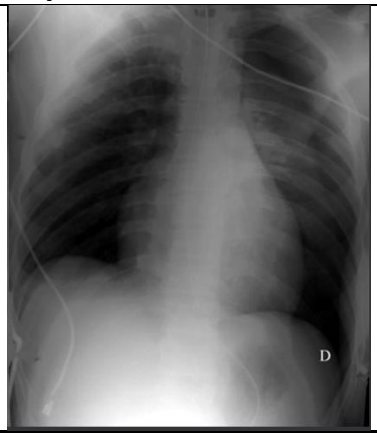
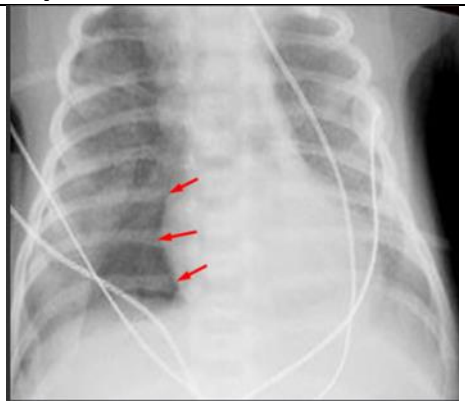
Open pneumothorax => Air moves freely during respiration
Closed pneumothorax => No movement of air occurs.
Tension pneumothorax => Valve like mechanism = air in not out.

❖ Pneumothorax in Supine Position

1) Sharp delineation of mediastinal contour
2) Visibility of medial diaphragm under cardiac silhouette
3) Deep sulcus sign = anterior costophrenic angle sharply delineated.

4) Sharply outlined diaphragm
5) Double diaphragm sign

6) Sharply delineated pericardial fat
7) Sharply delineated cardiac apex.

Tension Pneumothorax	**White pleural line**
	
Deep sulcus sign in Supine vew	**Hyperlucent right cardiac border in supine veiw**
	

Malignant Pleural Mesothelioma (MPM)

- Most common primary malignancy of the pleura.
- 2nd most common malignancy of the pleura after metastatic disease
- Asbestos exposure in approximately 80% of patients.
- Latency period = 20 – 50y
- The patient prognosis is poor.
- Age = 50 – 70y
- Sex: M > F = 4:1

TNM Staging

Primary tumor (T)

T1b: Tumor involving the ipsilateral parietal pleura (mediastinal and diaphragmatic pleura) + visceral pleura

T2: Tumor involving ipsilateral pleural surfaces and any of the following structures:
- Diaphragm
- Interlobar fissures
- Lung parenchyma

T3: Tumor involving ipsilateral pleural surfaces + any of the following structures:
- Endothoracic fascia
- Mediastinal fat
- Single focus extending into the chest wall
- Pericardium (nontransmural)

T4: Tumor involving ipsilateral pleural surfaces + any of the following structures:
- Diffuse or multiple chest wall masses
- Transdiaphragmatic extension
- Direct extension to the contralateral pleura
- Direct extension into the mediastinal organs or spine
- Pericardium (transmural)

Lymph nodes (N)

N0: No regional LNs

N1: Ipsilateral bronchopulmonary or hilar lymph nodes

N2: Subcarinal or ipsilateral mediastinal lymph nodes, including ipsilateral internal mammary and peridiaphragmatic lymph nodes

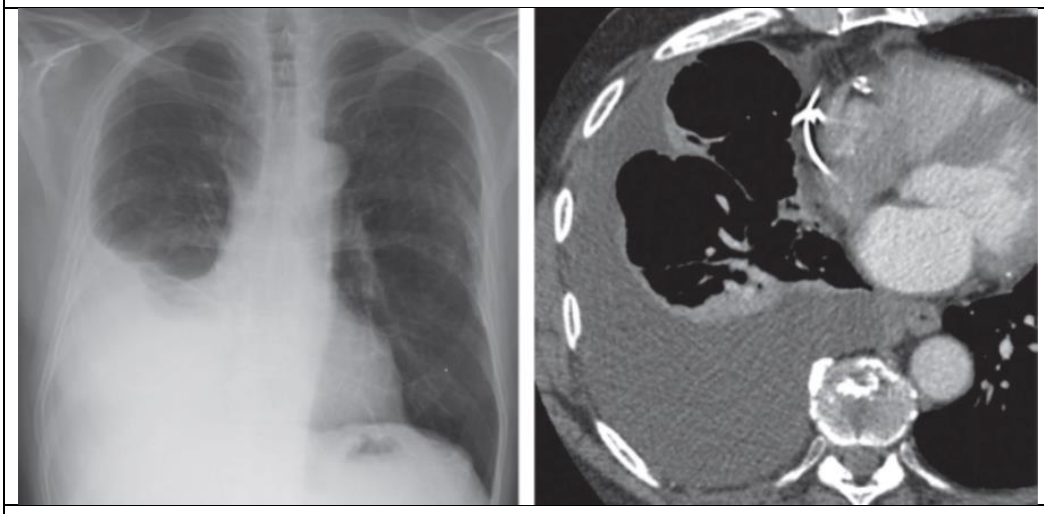
N3: Contralateral mediastinal, contralateral internal mammary, or ipsilateral or contralateral supraclavicular LNs

Metastases (M)

M0: No distant metastases

M1: Distant metastases

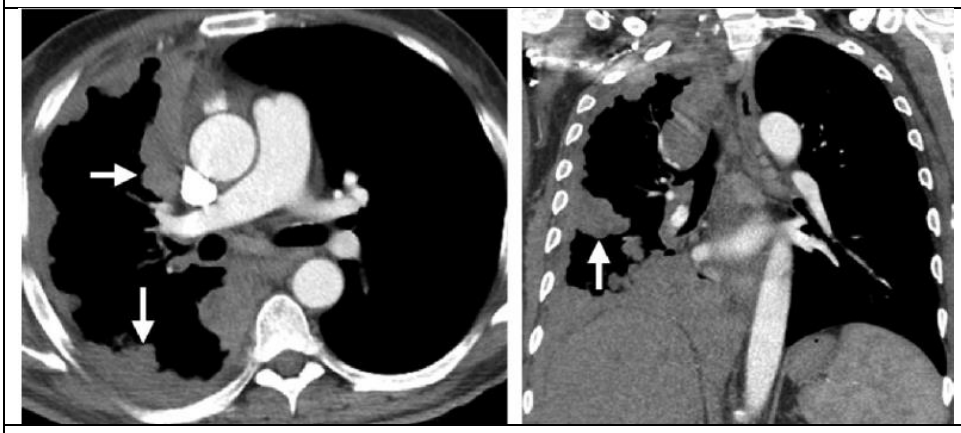
Pleural effusion



Pleural thickening:

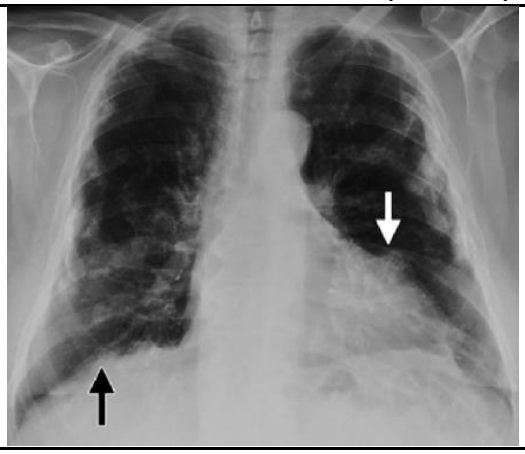
Irregular, nodular, circumferential, > 1cm

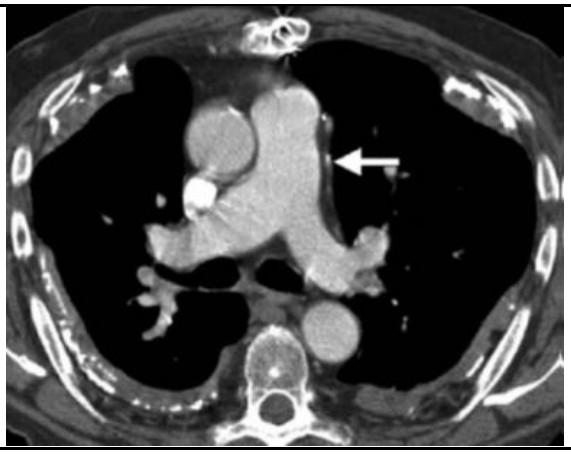
Fissural extension



Asbestos-related pleural disease

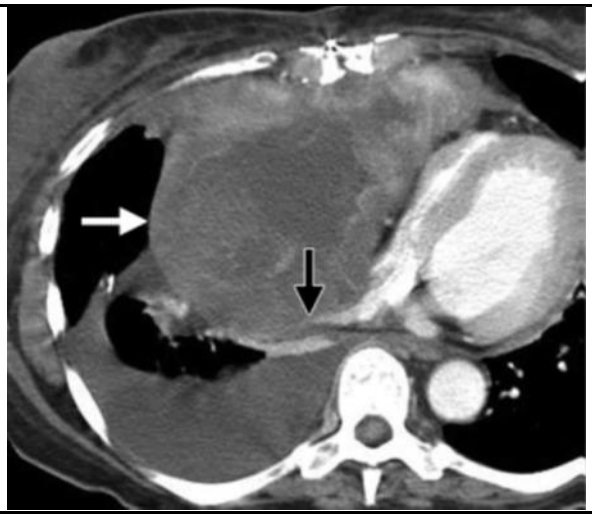
Calcified and noncalcified pleural plaques

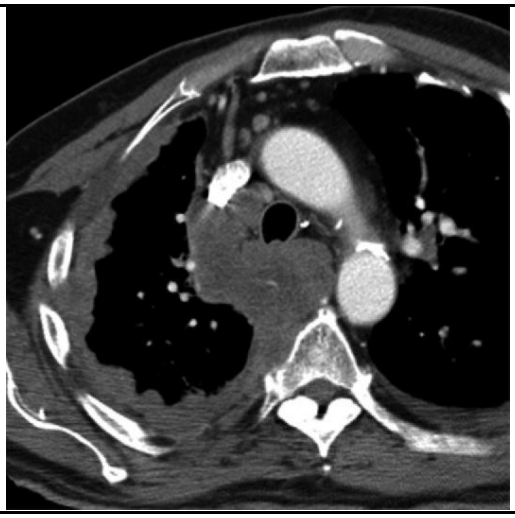




Mediastinal Invasion:

- Loss of fat and tissue planes between mediastinal structures.
- Encasement > 50% of the circumference of the trachea or esophagus or vascular structures.



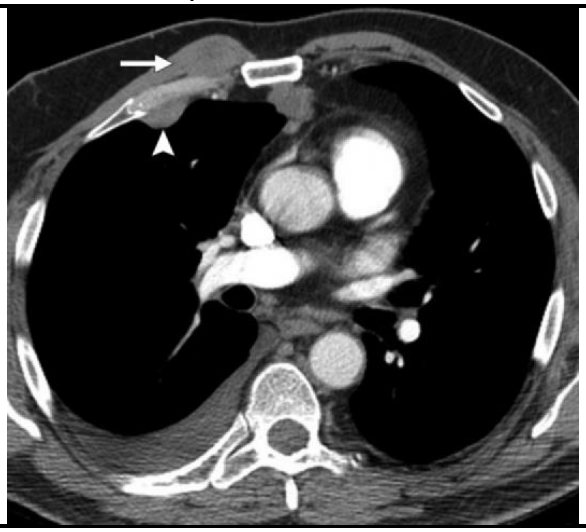


Pericardial invasion:

- May be nontransmural or transmural
- Pericardial effusion, thickening, nodules and masses, and/or infiltrating soft tissue

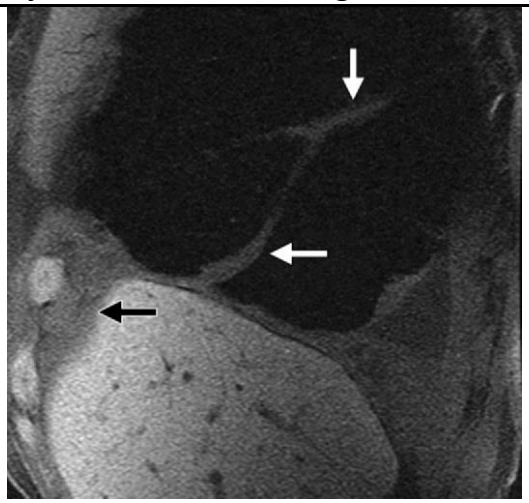
Chest Wall Invasion:

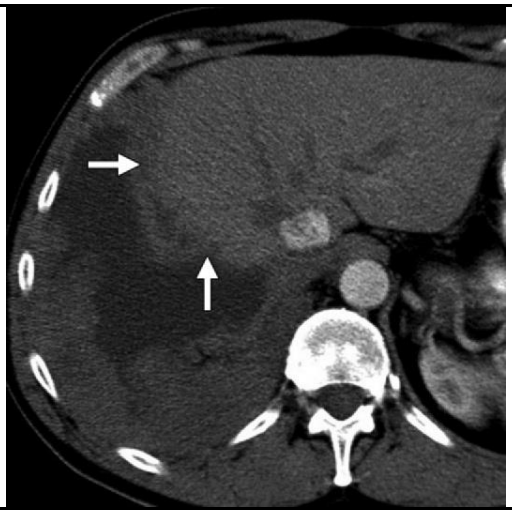
- ✓ Loss of normal extrapleural fat planes
- ✓ Invasion of intercostal muscles
- ✓ Rib displacement, or osseous destruction



Transdiaphragmatic Extension:

Loss of fat plane between the inferior surface of the diaphragm and the adjacent abdominal organs





LN Metastasis

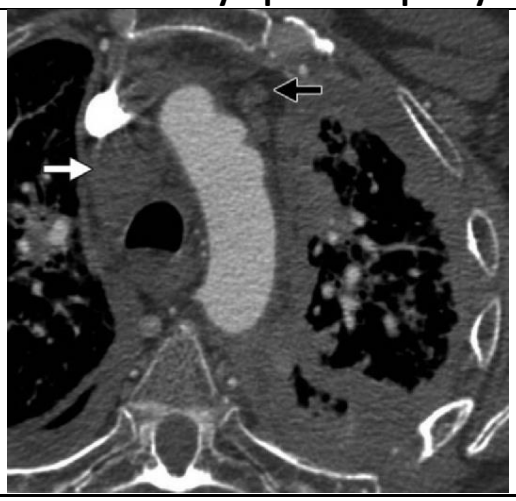
Mediastinal lymph nodes ≥10 mm

Pulmonary Metastases:

Pulmonary nodules / masses

Lymphangitis carcinomatosis = thickening and nodularity of interlobular septa

Mediastinal Lymphadenopathy



Lymphangitis Carcinomatosis

Pleural Metastasis

Pleural metastases are the most common malignancy of the pleura

The most common primary tumors to metastasize to the pleura are:

- Lung cancer (40%),
- Breast cancer (20%)
- Lymphoma (10%)
- Ovarian cancer
- Gastric cancer

Radiologically may be indistinguishable from MPM:

- ✓ Pleural effusion
- ✓ Pleural thickening
- ✓ Pleural nodules or masses

Fibrous Tumor of the Pleura

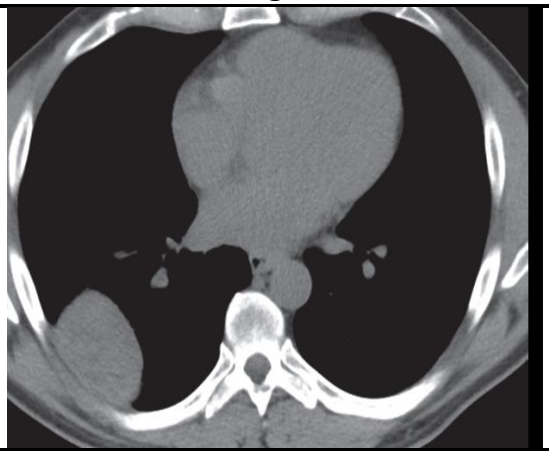
Low grade neoplasm - 20–30% of FTP are malignant
Not related to asbestosis
Originates from the submesothelial connective tissue
Arises from the visceral pleural surface

Associations (Uncommon 5%):

- ✓ Hypoglycemia
- ✓ Hypertrophic pulmonary osteoarthropathy

Radiological findings:

Pleural based lesions
Small lesions: Homogeneous + obtuse angle.
Large lesions: Heterogeneous + pedunculated + Acute angle

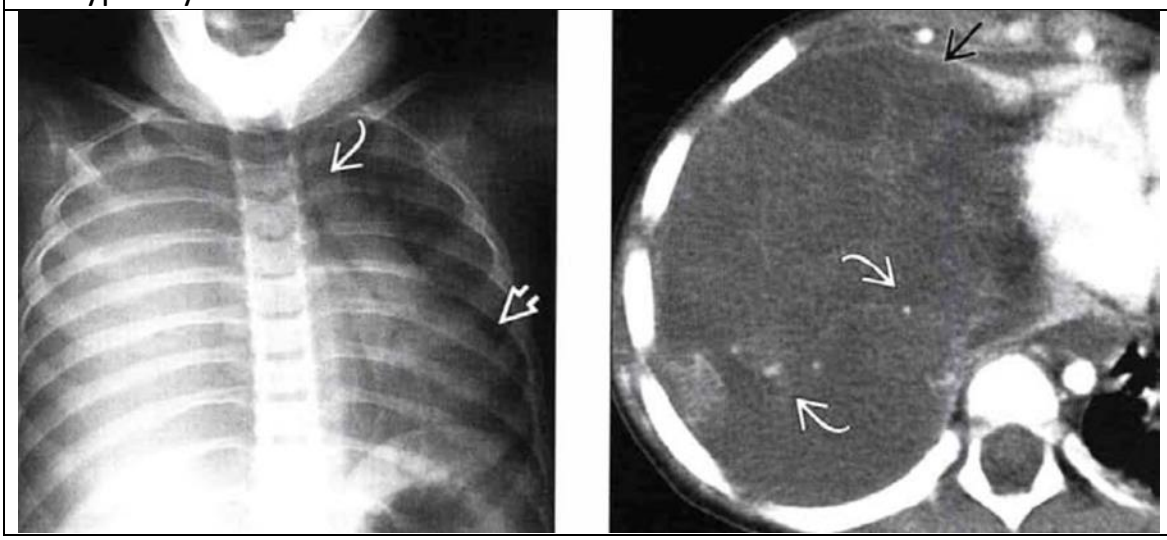


PLEUROPULMONARY BLASTOMA

Rare aggressive malignant primary neoplasm of the pleuropulmonary mesenchyme occurring in early childhood associated with poor prognosis.

- Type I: Purely cystic
- Type II: Cystic and solid
- Type Ill: Solid

- Opacified hemithorax with contralateral mediastinal shift
- Typically no chest wall invasion



Chest Wall Lesions

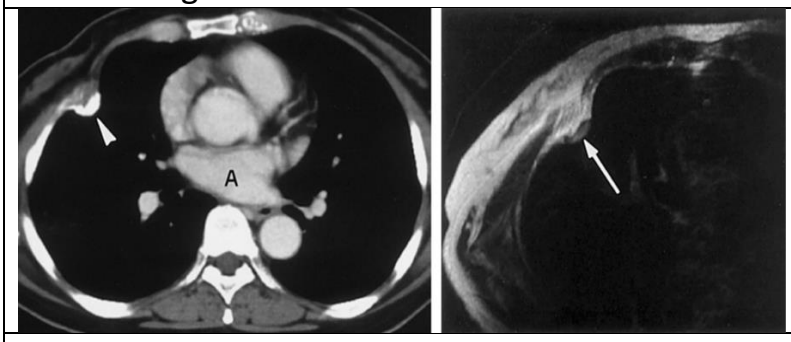
A) Osseous and Cartilaginous Tumors

1) Osteochondroma

- Ribs - Costochondral junction.
- Pedunculated osseous protuberances arising from the surface of the parent bone.
- Cartilaginous cap is of high signal on T2

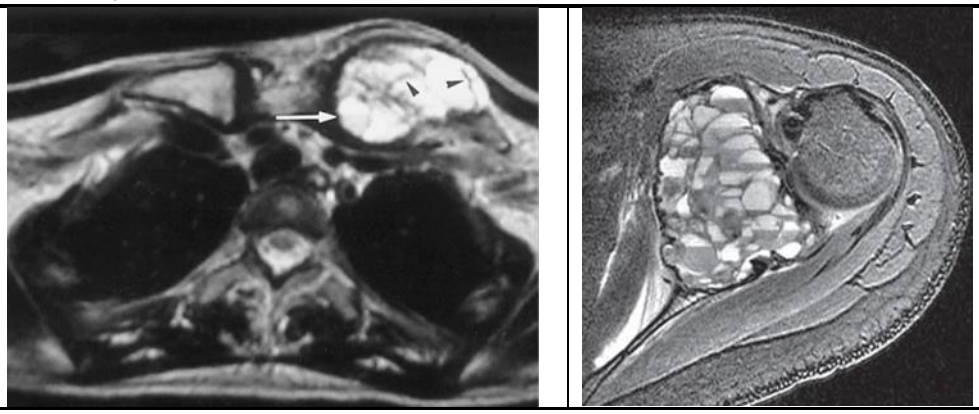
Complications:

- Fractures, osseous deformity
- Neural / vascular compression
- Bursa formation
- Malignant transformation.



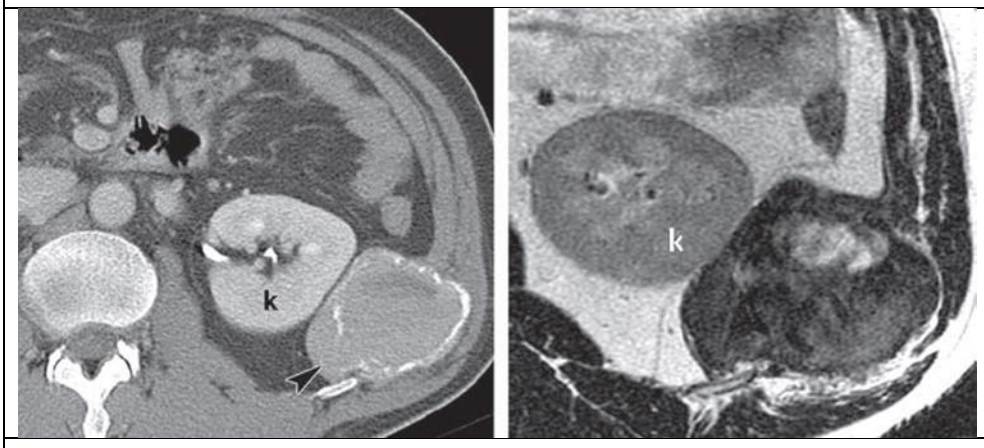
2) Aneurysmal Bone Cyst

- Well defined expansile multilocular cystic lesion
- Lobulated / Septated lesion
- Thin, well-defined rim of low signal intensity.
- Fluid/fluid level within the tumor



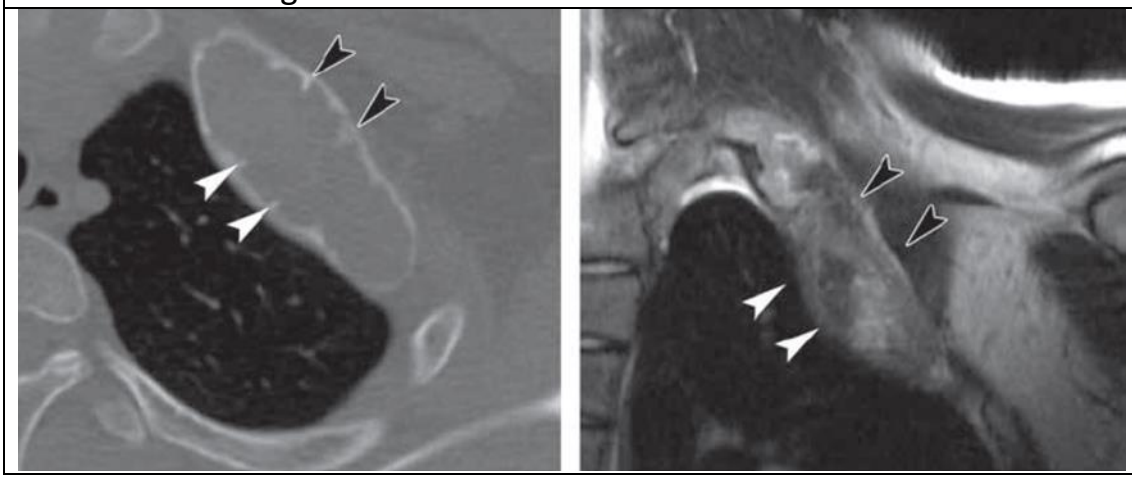
3) Giant Cell Tumor

Middle aged women
After closure of the epiphyses.
Flat bones of the chest wall, including the sternum, clavicle, and ribs.
Well defined expansile osteolytic lesion.
Cortical thinning out.
T1: Intermediate signal
T2: High signal
± Fluid-fluid levels



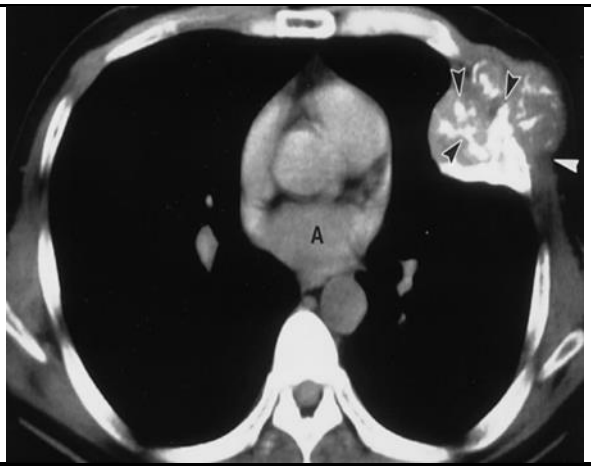
4) Fibrous Dysplasia

Well defined expansile lesion
Ground glass matrix
Endosteal scalloping – cortical thinning out
Bone remoddeling



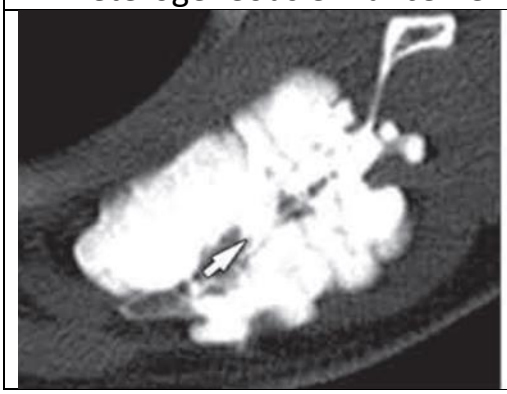
Chondrosarcoma

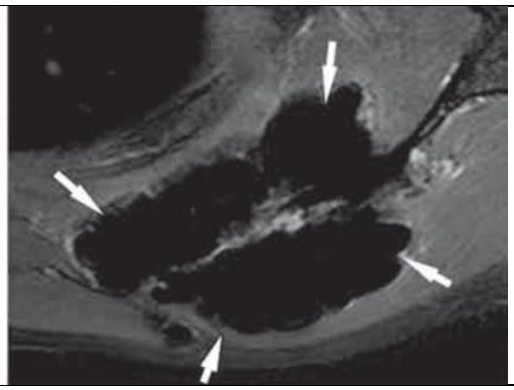
- 2nd most common primary malignant tumor of bone
- Arise de novo or secondarily from preexisting benign cartilage neoplasms.
- Age = 40 and 60 y.
- Chondroid matrix mineralization, with the characteristic ring-and-arc pattern of calcification best characterized by CT.
- Aggressive features such as soft tissue extension, cortical disruption, periosteal reaction.
- Deep endosteal scalloping help differentiate chondrosarcomas from benign cartilage neoplasms such as enchondromas.



Osteosarcoma

- Young adults
- The rib, scapula, and clavicle are the most frequent sites of origin.
- Bony matrix
- Heterogeneous enhancement.

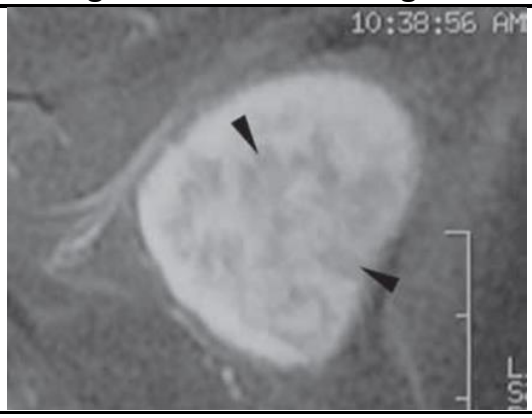


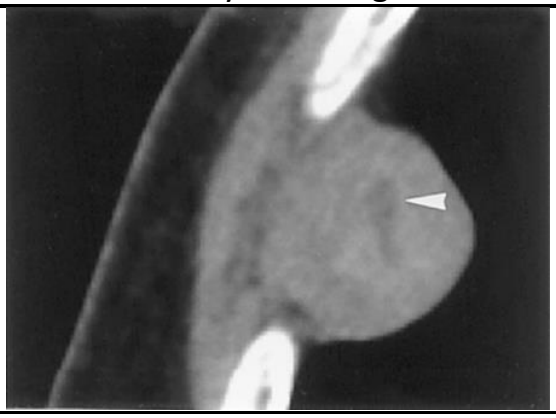


B) Peripheral Nerve Tumors

1) Schwannoma

- Well defined homogeneous mass.
- T1: Iso – hypointense
- T2: Marked hyperintense
- Small tumors => Intense homogenous enhancement
- Large tumors => Heterogeneous enhancement + cystic changes





2) Neurofibroma

Age = 20 and 30

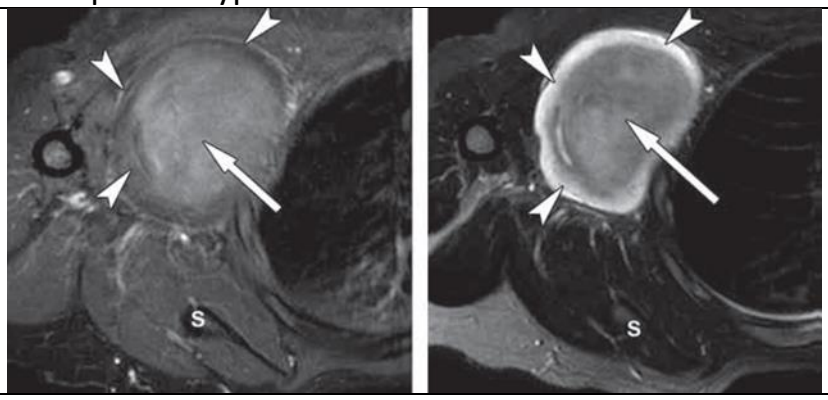
Associated with NF-1

T2: Target-like appearance =

- Central zone of low signal of a highly cellular component
- Peripheral zone of high signal of abundant stromal material.

T1+C: Target-like appearance =

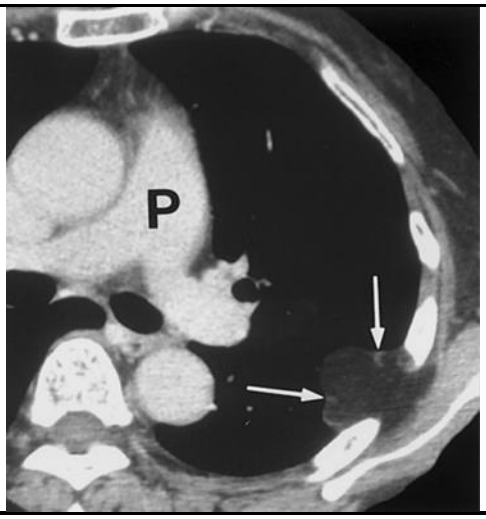
- Central marked enhancement
- Peripheral hypoenhancement



C- Adipose Tissue Tumors

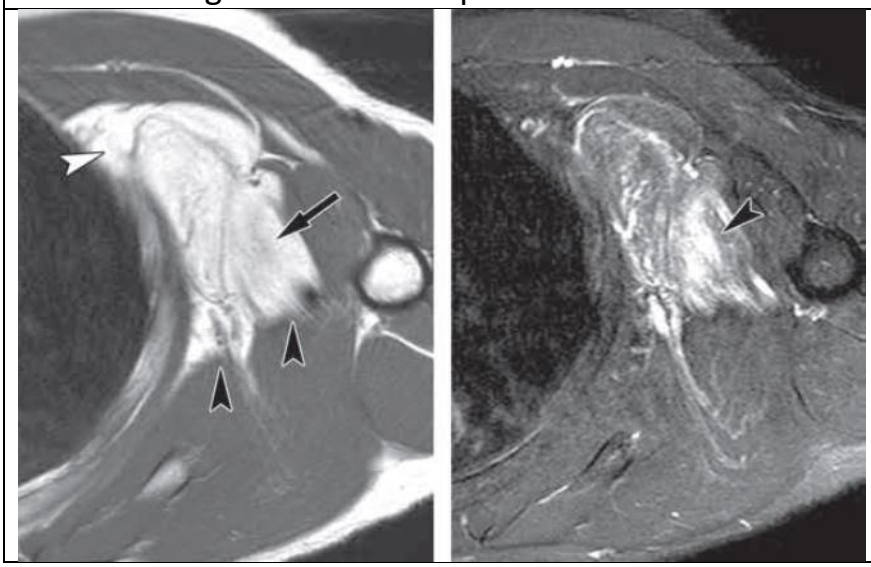
1) Lipoma

- well-circumscribed encapsulated mass
- Homogeneous density / signal
- CT: Fat density
- T1:Hyperintense signal
- T1 FS: Loss of high signal
- +C: No enhancement.



2) Liposarcoma

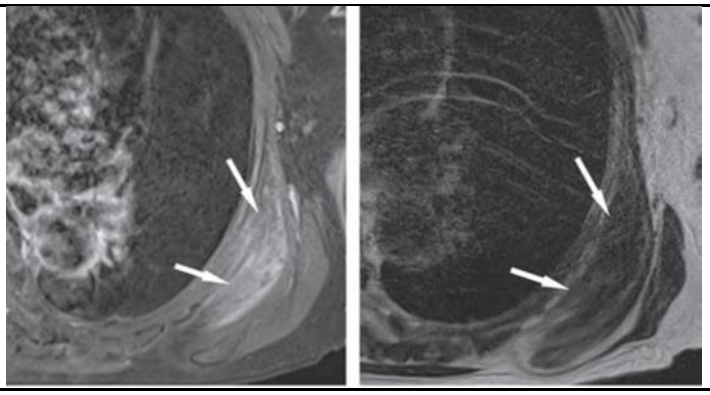
- Variable amounts of fat
- Enhancing soft tissue component



D- Muscular Tumors

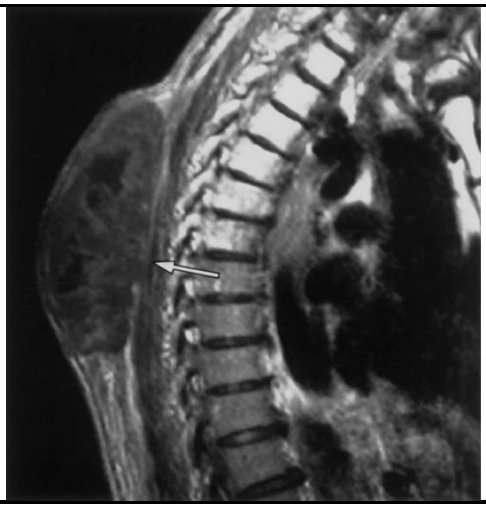
1) Elastofibroma Dorsi

Well defined crescent shaped soft-tissue lesion in the deep dorsal region between the thoracic wall and the lower third of the scapula.



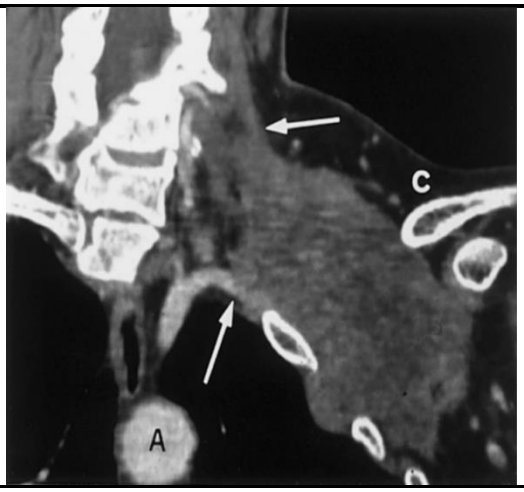
2) Leiomyosarcoma

M >> F
50–70 years.
Non-specific imaging features
Heterogeneous = Areas of necrotic or cystic change.
T1: Low signal
T2L High signal
Heterogeneous enhancement



3) Rhabdomyosarcoma

Young Adults
Histologic subtypes: embryonal, alveolar, and pleomorphic.
Non-specific heterogeneous mass



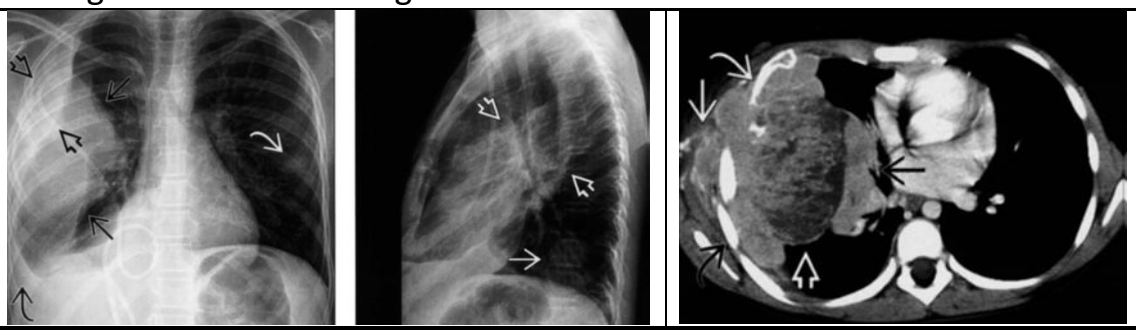
4) Malignant Fibrous Histiocytoma (MFH)

Old adults.
Nonspecific heterogeneously enhancing mass

5) Ewing Sarcoma Family = Peripheral Primitive Neuroectodermal Tumor (pPNET)

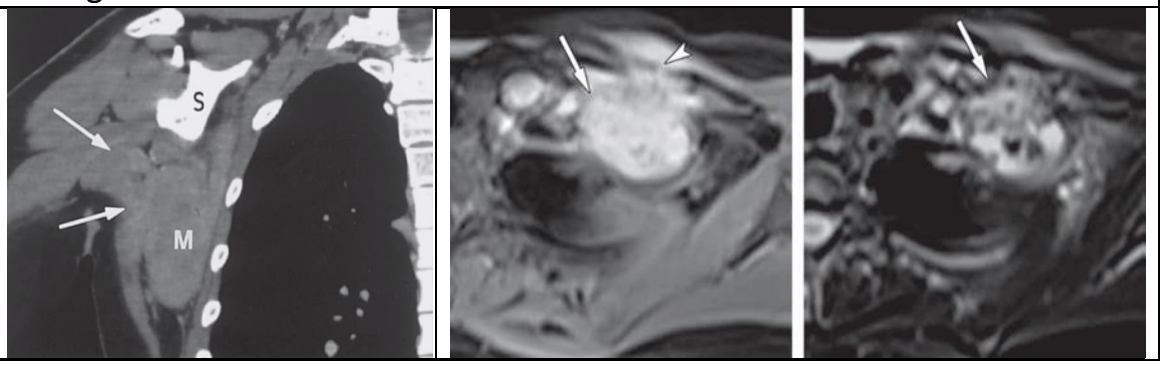
- Ewing sarcoma
- Peripheral primitive neuroectodermal tumor (pPNET)
- Neuroepithelioma
- Atypical Ewing sarcoma
- Askin tumor

- Children and adolescents.
- Tumors usually occur in the rib, scapula, clavicle, or sternum
- occasionally have an extraskeletal site of origin.
- Large ill-defined heterogeneous mass



7) Fibromatosis

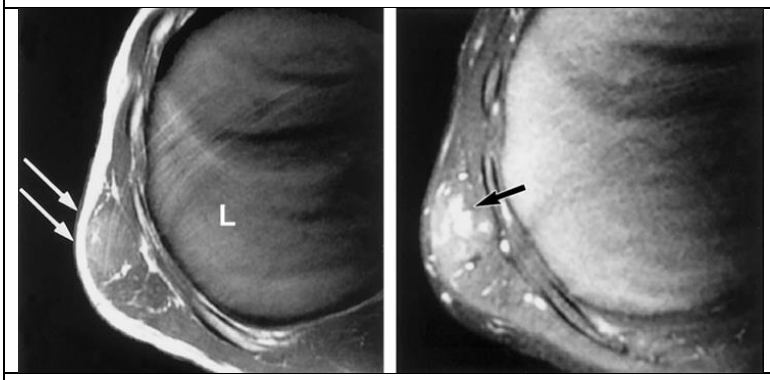
Adolescents and young adults
Shoulder involvement being the most common complication.
Low signal on T1 & T2



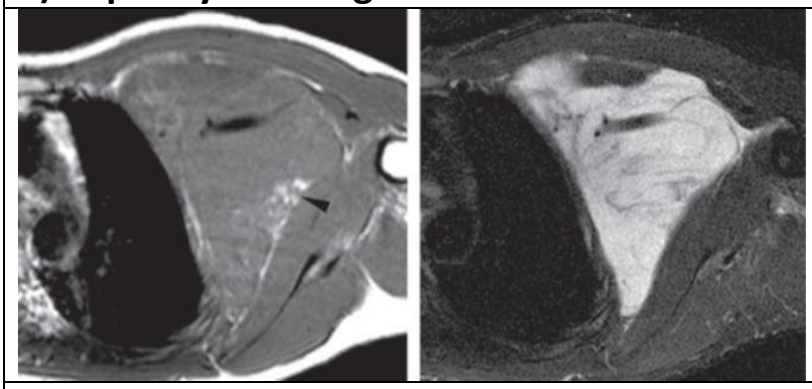
E- Vascular Malfromations

1) Cavernous Hemangioma

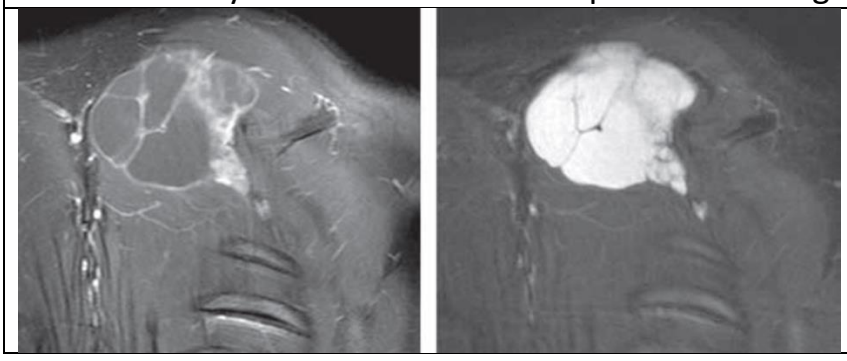
Cutaneous in location
Large, poorly circumscribed soft tissue mass.
CT: phleboliths
T2: Marked high signal
+C: Intense contrast enhancement.



2) Capillary Hemangioma

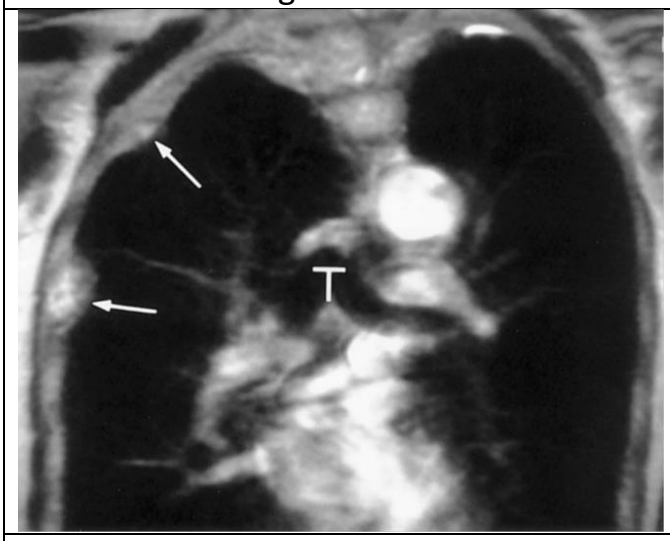


Multilocular cystic lesion + Internal septations + Marginal enhancement



4) Glomus Tumor

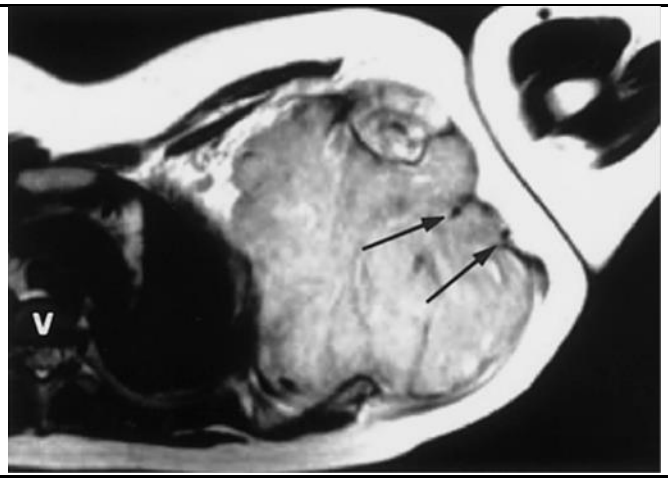
Intense enhancing sift tissue tumors



5) Angiosarcoma

Chronic lymphedema is the most widely recognized factor predisposing individuals to angiosarcoma

Associated with breast radiation therapy.



F- Hematologic Malignancies

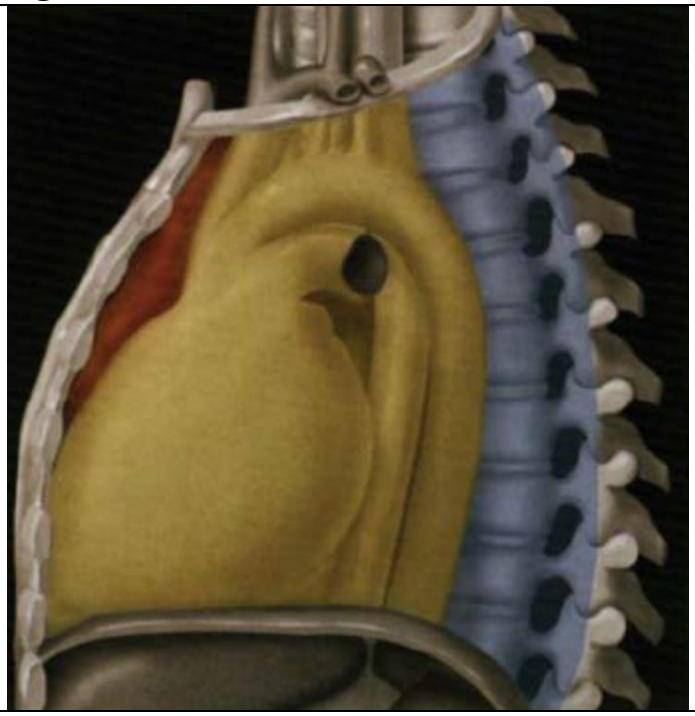
Lymphoma – leukemia – Metastasis – Multiple Myeloma

G- Cutaneous Lesions

1) **Epidermal Inclusion Cyst**
2) **Dermatofibrosarcoma Protuberans (DFSP)**
3) **Pilomatricoma**

Mediastinum Anatomy

The mediastinum is divided into 3 compartments to aid in the differential diagnosis



I) Anterior Mediastinum

- is the space between the sternum and pericardium inferiorly and ascending aorta and brachiocephalic vessels superiorly.

• The anterior mediastinum is divided into 2 compartments:

A) Prevascular compartment = Superior Anterior Mediastinum

- **Contents of prevascular space:**
  - Thymus.
  - Lymph nodes.
  - Enlarged thyroid gland.

B) Precardiac compartment = Inferior Anterior Mediastinum

- Potential space.

II) Middle mediastinum

- The anterior border is the anterior pericardium
- The posterior borders is posterior pericardium and posterior tracheal wall.

- **<u>Contents of middle mediastinum include:</u>**
  - Heart and pericardium.
  - Ascending aorta and aortic arch.
  - Great vessels including SVC, IVC, pulmonary arteries and veins, and brachiocephalic vessels.
  - Trachea and bronchi.
  - Lymph nodes.
  - Phrenic, vagus, and recurrent laryngeal nerves

III) Posterior mediastinum

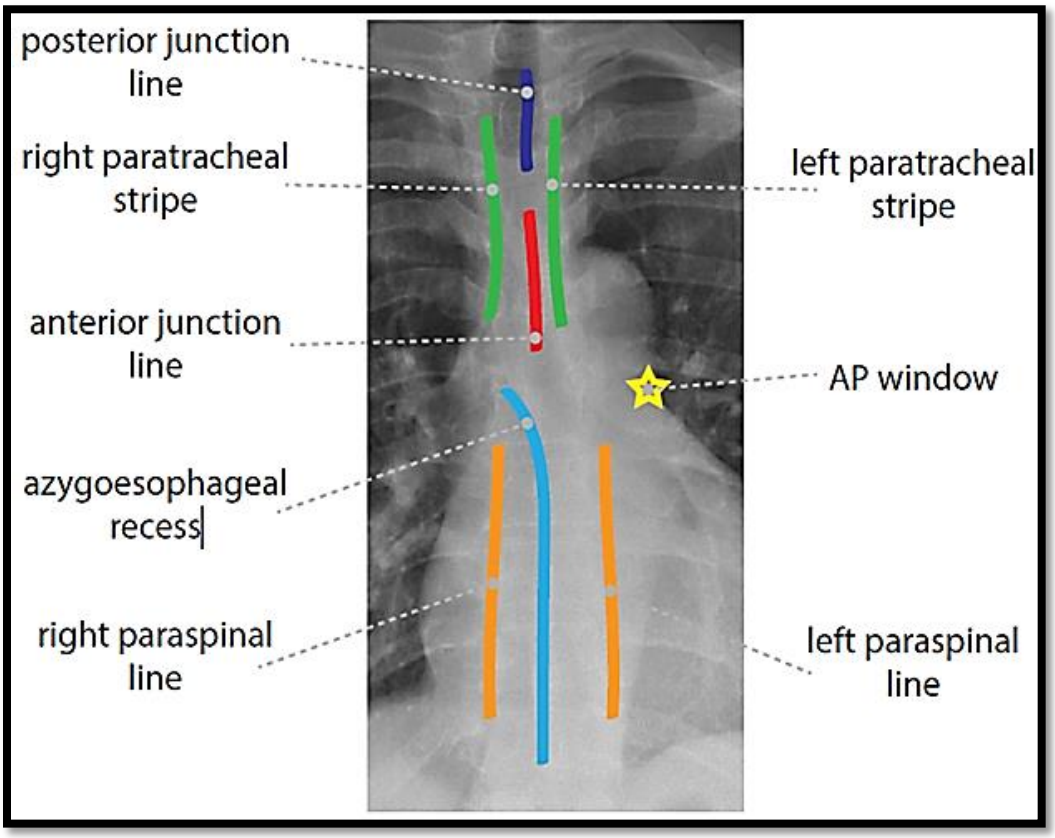
- The anterior border is the posterior trachea and posterior pericardium.
- The posterior border is the anterior aspect of the vertebral bodies.

- **<u>The contents of the posterior mediastinum include:</u>**
  - Esophagus.
  - Descending thoracic aorta.
  - Azygos and hemiazygos veins.
  - Thoracic duct.
  - Vagus nerves.
  - Lymph nodes

Mediastinal / Pleural Lines, Stripes, Interfaces

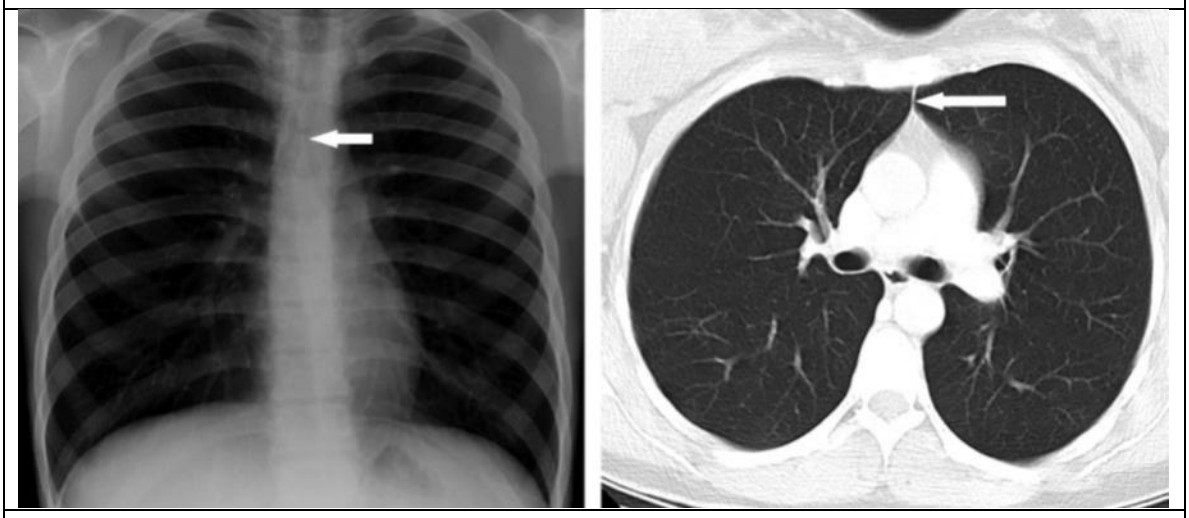
- Interfaces between anatomic structures in the lungs, mediastinum, and pleura may be displaced or thickened in the presence of a mediastinal mass or abnormality.

- A “line” is a thin interface formed by tissue (typically <1 mm in thickness) with air on both sides.
- A “stripe” is a thicker interface formed between air and adjacent soft tissue.
- An “interface” is formed by the contact of two soft tissue structures of different densities.



Anterior junction line:

- Formed by four layers of pleura (parietal and visceral pleura of each lung) at the anterior junction of the right and left lungs.
- CXR PA: Vertical line projecting over the superior 2/3 of the sternum.
- Abnormal convexity or displacement of this line suggests an anterior mediastinal mass.

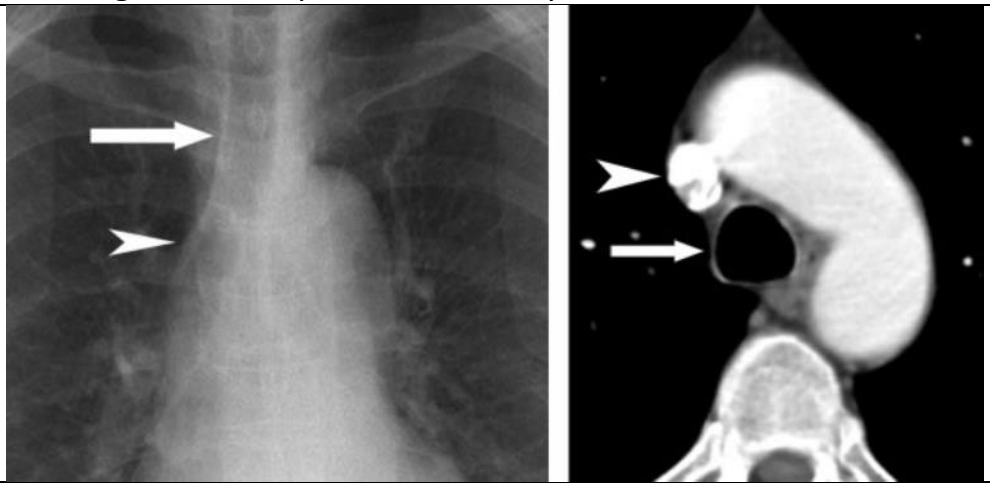


Posterior Junction Line:

- Formed by four layers of pleura (parietal and visceral pleura of each lung), at the posterior junction of the right and left lungs.
- CXR PA: Vertical line projecting through the trachea, more superior than the anterior junction line, above the clavicles.
- Abnormal convexity or displacement of this line suggests a posterior mediastinal mass or aortic aneurysm.

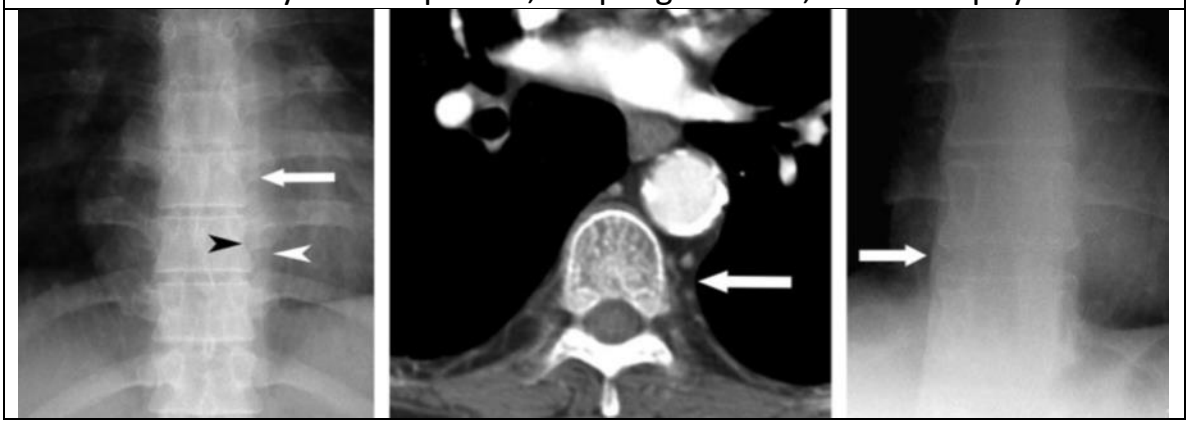
Right and left paratracheal stripes:

- Formed by two layers of pleura where the medial aspect of each lung abuts the lateral wall of the trachea and intervening mediastinal fat.
- The right paratracheal stripe is the most commonly seen of these landmarks, seen in up to 97% of normal CXR PA
- Thickening of the right paratracheal stripe is most commonly due to pleural thickening, although a paratracheal or tracheal mass (including adenopathy or thyroid or tracheal neoplasm).
- Thickening of the left paratracheal stripe has a similar differential.



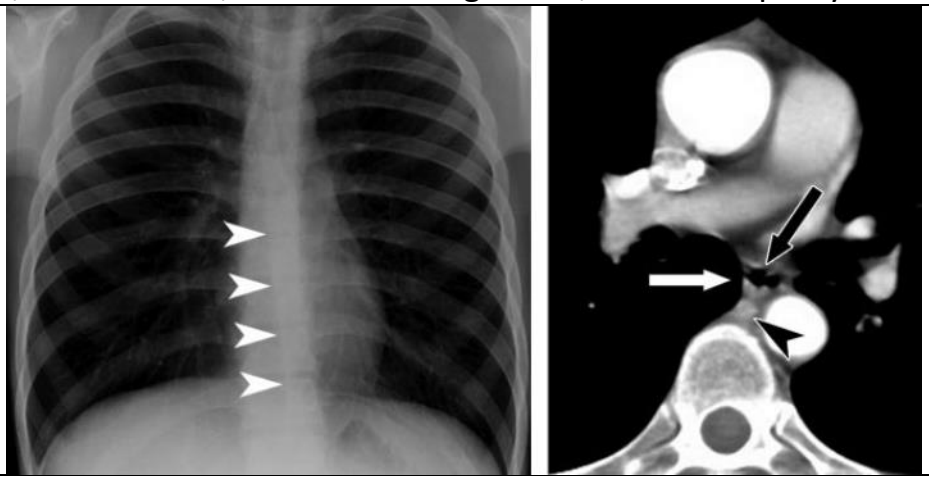
Right and Left Paraspinal lines

- Formed by 2 layers of pleura abutting the posterior mediastinum.
- In contrast to the posterior junction line, the paraspinal lines are located inferiorly in the thorax, typically from the 8th through 12th ribs.
- A paraspinal line abnormality suggests a posterior mediastinal mass lesion, including hematoma, neurogenic tumor, aortic aneurysm, extramedullary hematopoiesis, esophageal mass, and osteophyte.



Azygoesophageal Recess

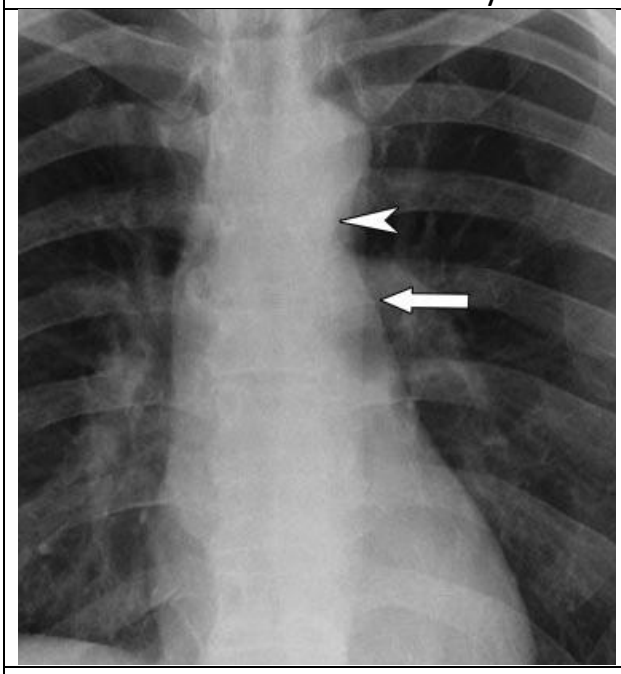
- Formed by the contact of the posteromedial right lower lobe and the retrocardiac mediastinum.
- The azygoesophageal recess extends from the subcarinal region to the diaphragm inferiorly.
- Distortion of the azygoesophageal recess may be due to esophageal mass, hiatal hernia, left atrial enlargement, and adenopathy.



Aortopulmonary (AP) Window

- is a mediastinal space nestled underneath the aortic arch (which forms the superior, anterior, and posterior boundaries) and the top of the pulmonary artery.
- The medial border of the AP window is formed by the esophagus, trachea, and left mainstem bronchus.
- CXR PA: AP window is a shallow concave contour below the aortic knob and above the pulmonary artery.
- Abnormal convexity (outwards bulging) of the AP window suggests a mass, including:
    - Lymph nodes: Adenopathy is the most common cause of an AP window abnormality.
    - Left phrenic nerve: Injury may cause paralysis of the left hemidiaphragm.
    - Recurrent laryngeal nerve: The AP window should be carefully evaluated in new-onset hoarseness, especially if associated with diaphragmatic paralysis.
    - Left vagus nerve.
    - Ligamentum arteriosum.

- Left bronchial arteries.
- Thoracic aortic aneurysm



Retrosternal clear space

- is a normal area of lucency posterior to the sternum seen on the lateral radiograph only.
- It correlates to the prevascular space on CT.
- Obliteration of the retrosternal clear space suggests an anterior mediastinal mass, right ventricular dilation, or pulmonary artery enlargement.
- Increase in the retrosternal clear space can be seen in emphysema.

Posterior Tracheal Stripe

- is the only interface seen on the lateral CXR
- Representing the interface of the posterior wall of the trachea with the two pleural layers of the medial right lung.

I- Anterior Mediastinal Mass

A) Prevascular Space (Superior Anterior Mediastinum)

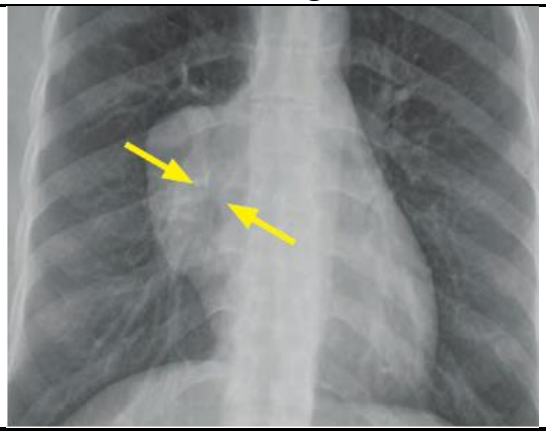
Soft Tissue	Cyst	Fat
• Lymphoma (1st common) • Germ cell Tumors (2nd common in children) • Thymoma (2nd common in adults) • ALL Relapse • Thymic Hyperplasia • Thyroid	• Thymic cyst • Lymphangioma • Abscess	• Teratoma • Thymiolipoma • Lipomatosis

CXR PA:

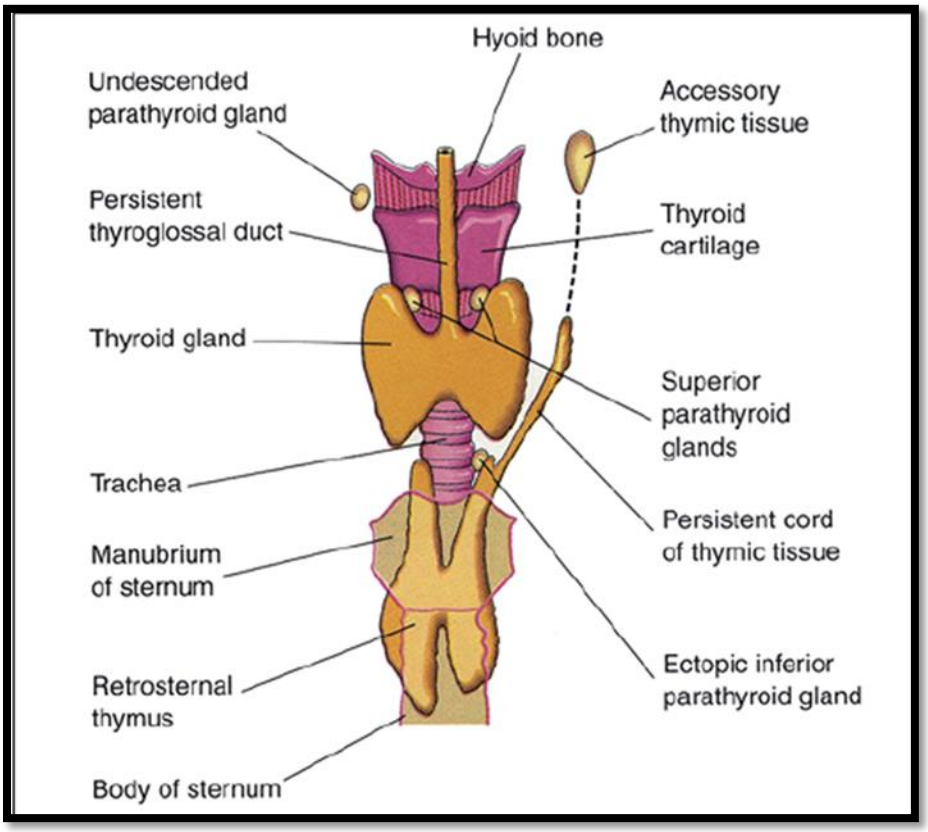
- The hilum overlay sign = Hilar vessels are visualized through the mass.
    - Not middle mediastinum mass.
- Preserved descending aorta and paravertebral lines
    - Not posterior mediastinal mass.
- Obliterated anterior junction line
    - Anterior mediastinal mass

CXR Lateral

- Obliteration of the retrosternal clear space
    - Direct sign of anterior mediastinal location.



Thymus Gland



- is a lymphatic organ that plays a vital role in the development and maturation of the immune system during childhood, specifically T and B cells.

Embryologically:

- **During the 6th gestational week**, thymic primordia arise from 3rd ,4th pharyngeal pouches.
- **During the 7th week,** the bud-like thymic primordia elongate and become cylindrical, forming thymopharyngeal ducts, and migrate caudally and medially to their final destination in the anterior mediastinum.
- **During the 8th gestational week,** the thymic primordia fuse at their lower poles.
- **During the 10th week** small lymphoid cells migrate from fetal liver tissue and bone marrow into the primordia, causing thymic lobulation.
- **During the 14th to 16th weeks,** the thymus further differentiates into cortical and medullary component

Anatomy:

- Located in the anterior mediastinum.
- It overlies the pericardium, aortic arch, left innominate vein, and trachea.
- may extend superiorly to the lower pole of the thyroid
- may extend inferiorly to the diaphragm.
- Attached to the thyroid by the thyrothymic ligament

Size:

- CT: The maximum thickness of the thymus is:
  - 1.8 cm <20 years
  - 1.3 cm >20 years
- The thickness is greater at MR imaging:
  - 1.5–2cm > 20 years

Shape of normal thymus:

Age < 5 y:

- Normal thymus quadrilateral shaped
- convex margins
- Soft tissue density (=muscles)

Age> 5 y:

- Thymus gradually becomes more triangular
- Thymic margins slowly straighten
- Soft tissue density (=muscles)

Age> 15 y

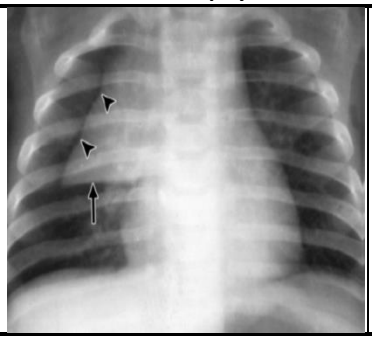
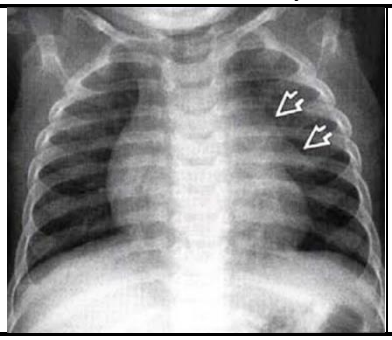
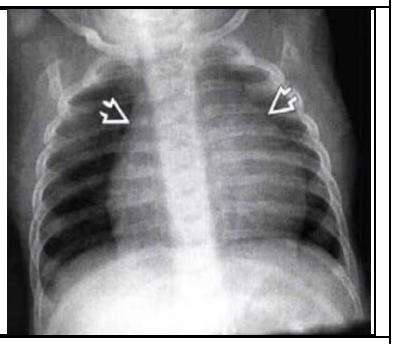
- Thymus is triangular
- Thymic margins should be straight or concave
- Fat density

Ectopic And Accessory Thymic Tissue

- Occur anywhere along the path of descent (thymopharyngeal duct) as the result of failure of descent, sequestration, or failure to involute.
  - Neck (32%)
  - Mediastinum (98%).
  - one or both lobes were found behind the thyroid (26%)

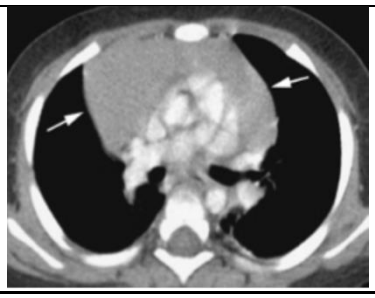
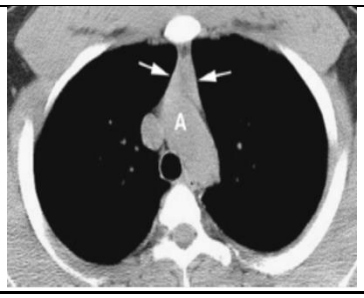
❖ CXR of Normal Thymus

- **Thymic wave sign**, a scalloped or wavy contour of the organ, is created by the impression of the anterior reflection of the ribs
- **Thymic sail sign**, a triangular, slightly convex right lobe of the thymus with a sharply demarcated base caused by the minor fissure

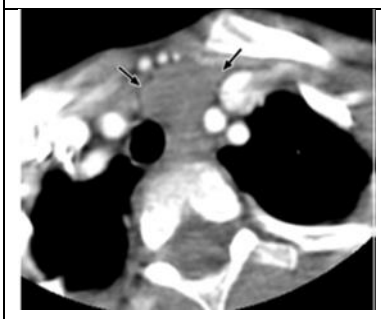
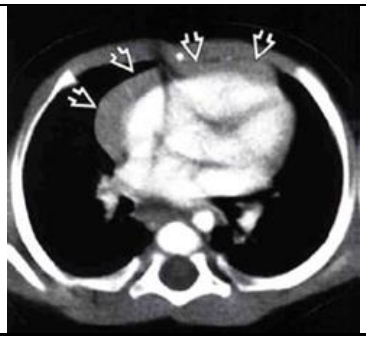
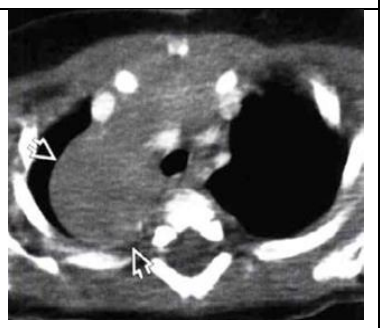
❖ CT of Nromal Thymus

Thymus < 5y	Thymus > 5y	Thymus >15y
Quadrilateral shape	Triangular shape	Triangular shape
Convex margins.	Straight margins	Straight margins
Soft tissue attenuation	Soft tissue attenuation	Fat attenuation

❖ Normal Anatomical Variants:

Cervical	Circumcardiac	Retrocaval

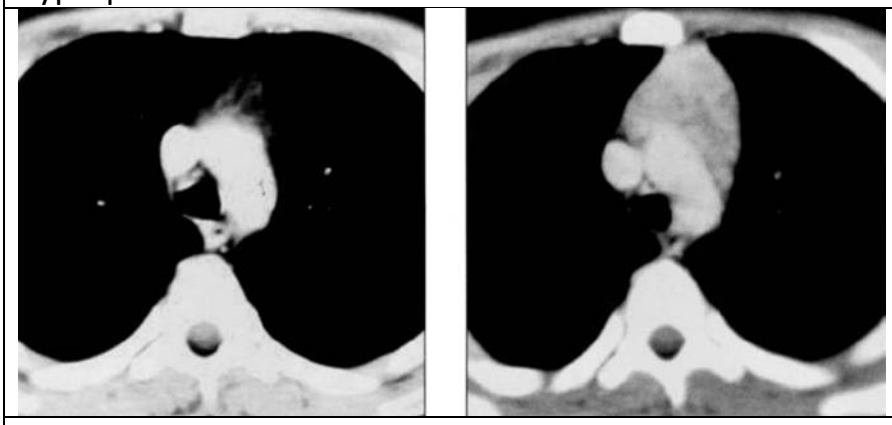
Thymic Hyperplasia

True thymic hyperplasia	Lymphoid thymic hyperplasia
○ Rebound hyperplasia to chemotherpay ○ Radiation therapy ○ Burns ○ Severe systemic stress	○ Myaesthenia gravis ○ Systemic lupus erythematosus (SLE) ○ Rheumatoid arthritis ○ Scleroderma ○ Graves disease

<u>Imaging Finding:</u>

Diffuse symmetric enlargement of the gland + Smooth contour

ALL patient 3 months after chemotherapy demonstrates thymic rebound hyperplasia.



Lymphoma

Most common anterior mediastinal mass in children

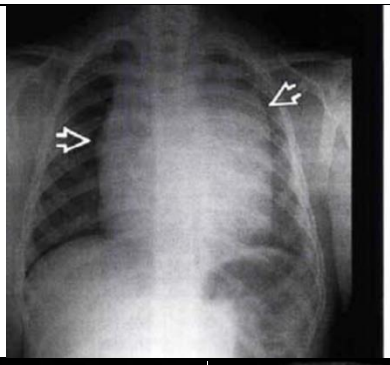
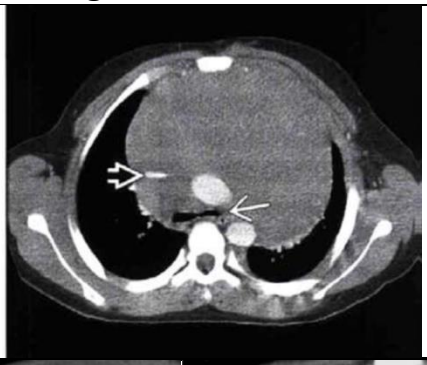
HD > NHL

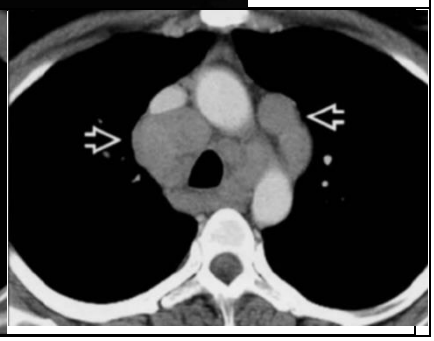
Hodgkin
- Stage I: Single lymph node group
- Stage II: 2 lymph node groups same side of diaphragm
- Stage III: Nodes on both sides of diaphragm
- Stage IV: Extranodal sites
  - A: Asymptomatic
  - B: Symptoms (20%) fever, night sweats, weight loss of 10%

<u>Imaging Findings:</u>

Superior mediastinal LNs including prevascular, AP window, and paratracheal LNs groups.

Anterior & middle mediastinal mass + Irregular nodular contour



Non-Lymphomatous Lymphadenopathy:

- **Eggshell calcification of LNs:**
  - Silicosis and coal workers pneumoconiosis
  - Less commonly in sarcoidosis

- **Low attenuation LNs:**
  - Active tuberculosis.
  - Fungal infection
  - Lymphoma
  - Metastatic disease.

- **Avid LN enhancement:**
  - Castleman disease
  - Sarcoidosis
  - Vascular metastases:
    - Renal cell carcinoma.
    - Thyroid carcinoma.
    - Lung carcinoma.
    - Sarcoma.
    - Melanoma.

❖ Castleman disease

- also known as angiofollicular lymph node hyperplasia
- is a cause of highly vascular thoracic lymph node enlargement, of uncertain etiology.
- Localized Castleman disease is seen in children or young adults.
  - Surgical resection is usually curative.
- Multicentric Castleman disease manifests in older patients or in association with AIDS.
- Multicentric disease often results in systemic illness including fever, anemia, and lymphoma.
  - It is typically treated with chemotherapy.
- The key imaging finding of Castleman disease is avidly enhancing adenopathy.

Thymoma

2nd most common mediastinal neoplasm in adults after lymphoma.
Location: Anterior mediastinum.
Age: > 40 years old.

Associations

- Myasthenia gravis : most common association
  - 10 - 20% of patients with myasthenia gravis have a thymoma
  - 30 - 50% of patients with a thymoma have myasthenia gravis
- Pure red cell aplasia
  - 50% of patients with pure red cell aplasia have a thymoma
  - 5% of patients with a thymoma have pure red cell aplasia
- Hypogammaglobulinaemia
  - 5% of patients with hypogammaglobulinaemia have a thymoma
  - 10% of patients with a thymoma have hypogammaglobulinaemia
- Systemic lupus erythematosus (SLE)
- Rheumatoid arthritis
- Non-thymic cancers

Masaoka Staging System:

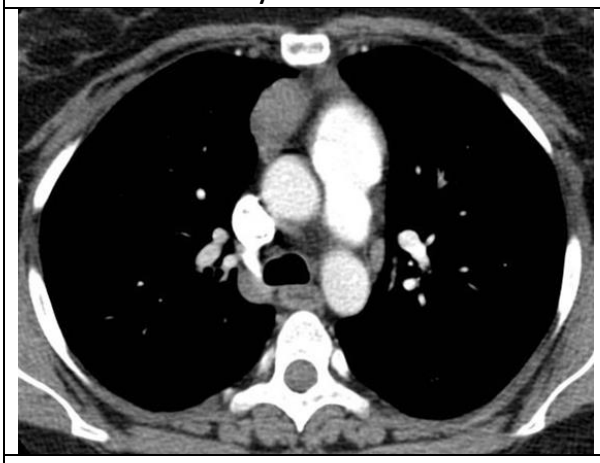
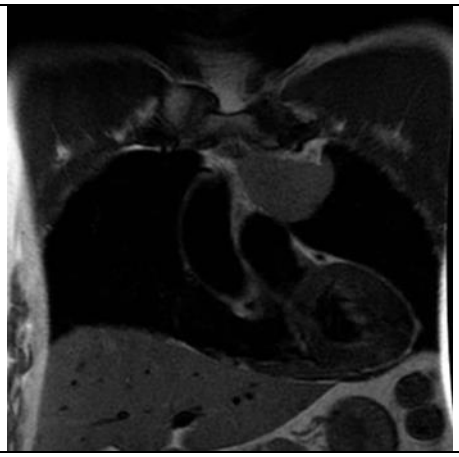
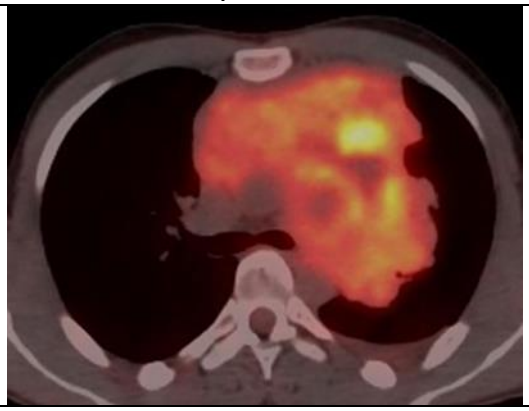
- Stage I : intact thymic capsule
- Stage II: capsular invasion into adjacent mediastinal fat or pleura.
- Stage III : macroscopic invasion into adjacent organs, vessels
- Stage IV :
  - IVa : dissemination in thoracic cavity (i.e pleural or pericardial implants)
  - IVb : distant metastases

Non-invasive Thymoma:

- Well defined masses of soft tissue attenuation
- Rounded, smooth or lobulated.
- Calcifications (20%)

Invasive Thymoma:

- Mediastinal fat invasion.
- Invasion of mediastinal structures including airways & great vessels.
- Invasion of chest wall.
- Mediastinal LNs enlargement may be present.
- Spread along pleural and pericardial surfaces = Drop metastases
- Elevation of hemidiaphragm is suggestive of phrenic nerve invasion

Non-inavsive thymoma = Well defined leison with smooth contour	
	
Calcification	Avid PET/CT uptake
	
Mediastinal fat invasion	Pleural drop metastasis

• Thymic Carcinoma

Unlike invasive thymoma, thymic carcinoma is histologically malignant,

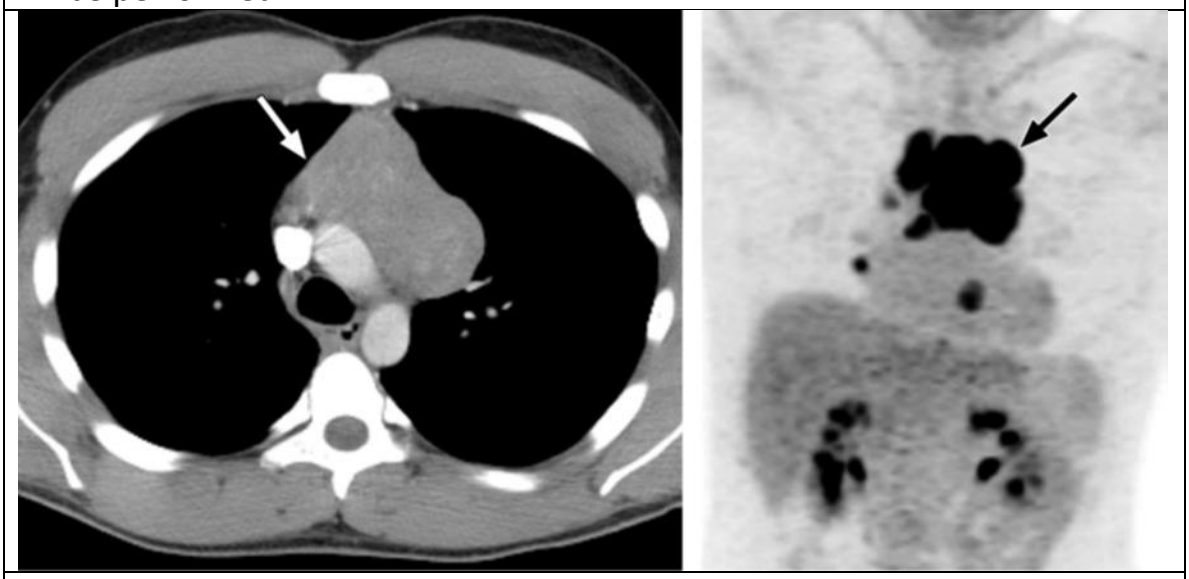
→ Very aggressive

→ Metastasizes hematogenously to lungs, liver, brain, and bone.

→ Prognosis is poor.

• Thymic Carcinoid

- Neural crest origin.
- 50% of thymic carcinoids are hormonally active, often secreting ACTH and causing Cushing syndrome.
- Associated with:
    - Multiple endocrine neoplasia (MEN) I and II.
- On imaging:
    - Thymic carcinoid is indistinguishable from thymoma and thymic carcinoma.
- If carcinoid is suspected, a preoperative Indium-111 Octreotide scan can be performed.



• Thymic Sarcoma

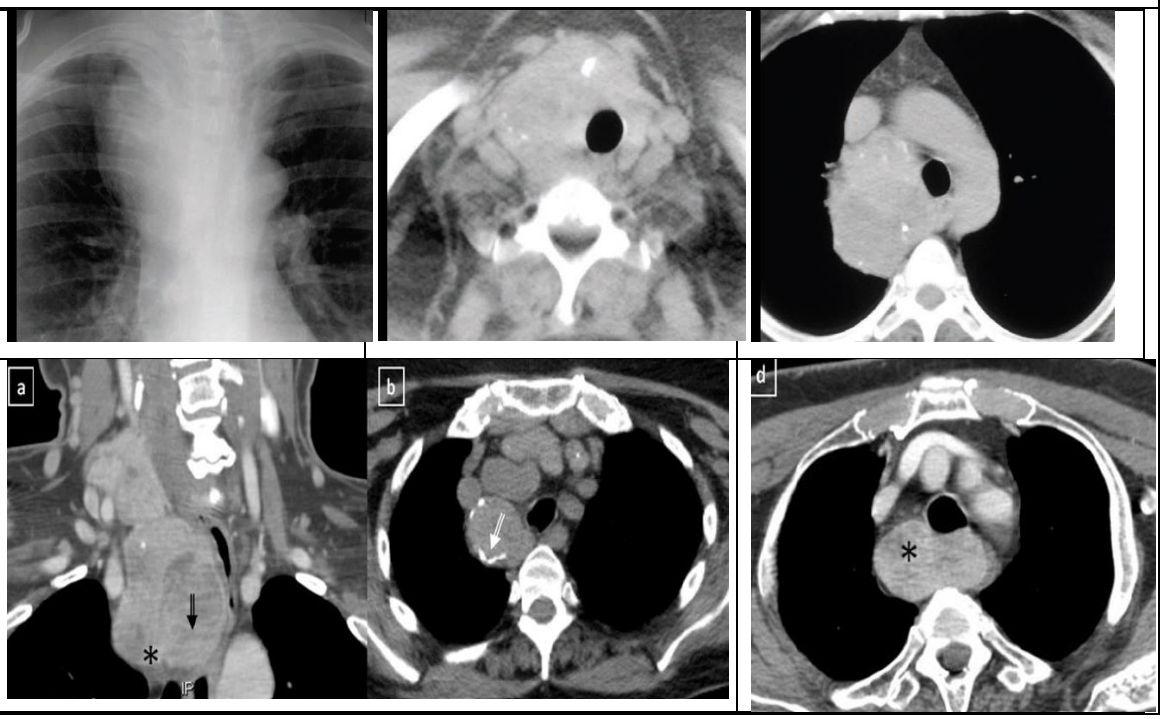
Extremely rare

Nonspecific appearance at cross-sectional imaging

❖ Retrosternal Goitre

Benign and malignant thyroid masses may extend into the mediastinum, including goiter, thyroid neoplasm, and an enlarged gland due to thyroiditis

- Superior mediastinal mass:
  - Retro-sternal location
  - Para-tracheal location
- Obscuration of superior border of the mass
- Cervicothoracic sign: continuity with the thyroid in the neck.
- High density + Heterogeneous + coarse calcifications
- Marked + prolonged + Heterogeneous enhancement
- Nodular well defined margins
- Contralateral tracheal shift

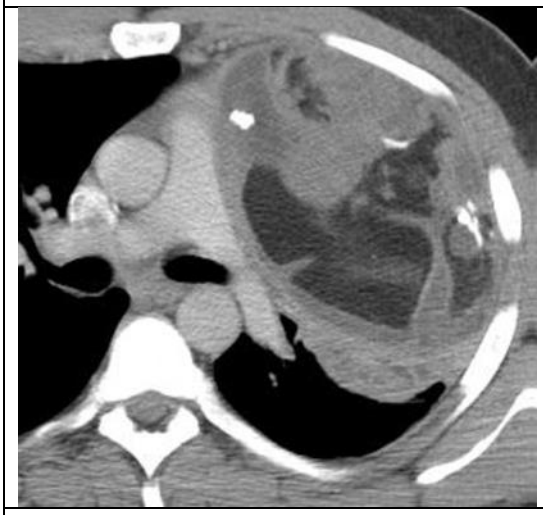


GERM CELL TUMORS

2nd most common cause of an anterior mediastinal mass in children.
Tumor derived from primordial germ cells that differentiate into embryonic and extraembryonic structures.
Age < 40y

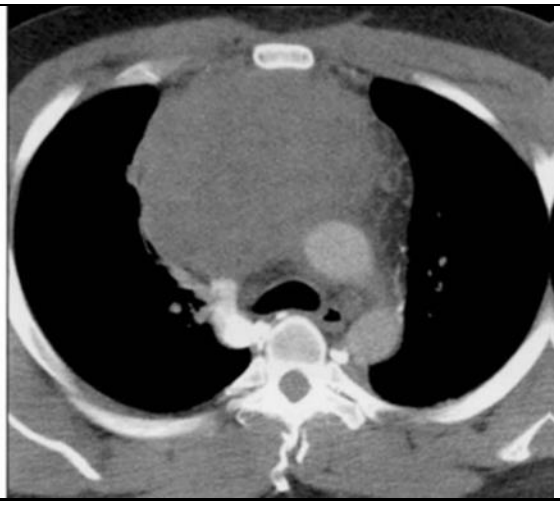
- Teratoma: Majority are benign and mature.
- Seminoma (10-20%): Germinoma and dysgerminoma
- Nonseminomatous germ cell tumor (NSGCT) (10-20%): Embryonal cell, endodermal sinus/yolk sac, choriocarcinoma, and mixed germ cell tumor

Teratoma = Fat + Fat/fluid + Soft tissue + Calcification



Choriocarcinoma
Elevated serum B-HCG

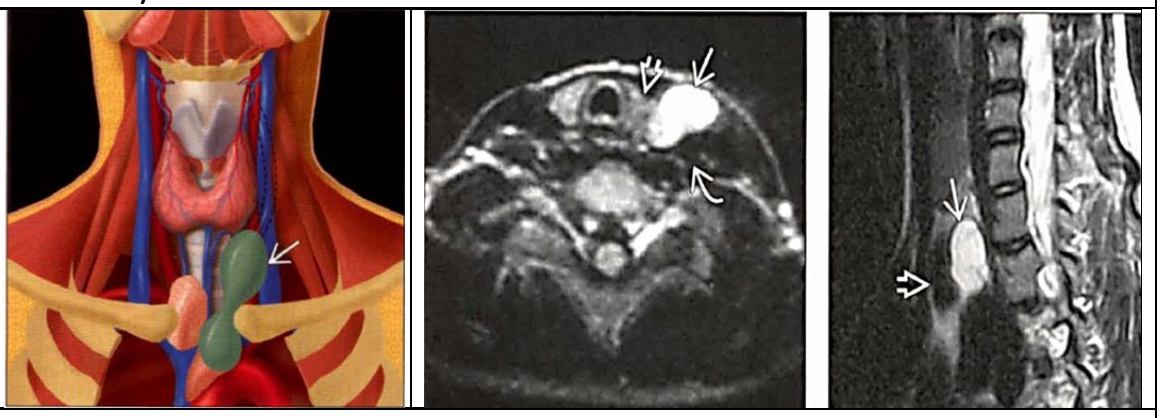
Semminoma



- **Cystic Lesions**

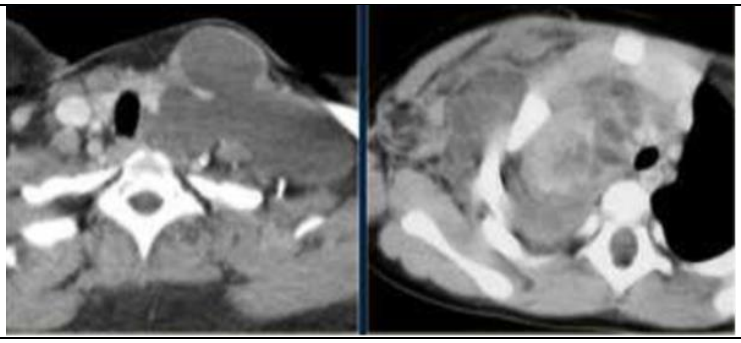
1) Thymic Cyst

Originate from embryonic remnants found along the thymopharyngeal duct
Extends from the upper neck to the anterior mediastinum.
Commonly in the lower neck



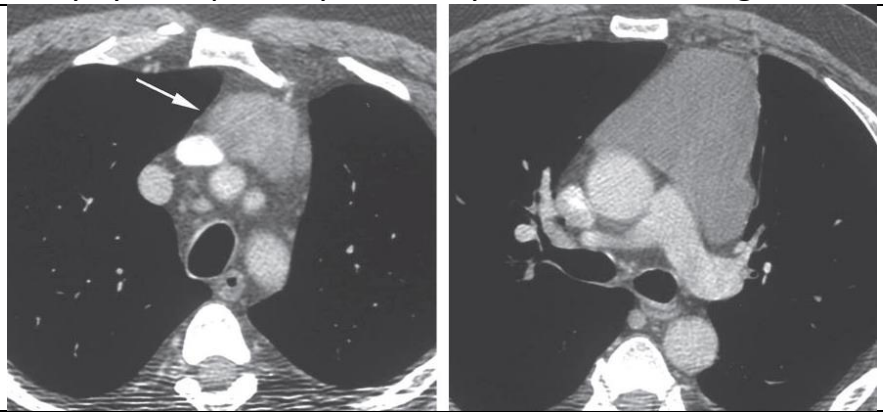
2) Lymphangioma = Cystic Hygroma:

- Age: Children.
- Multilocular cystic lesion ± Fluid / fluid level
- Extends to the neck



3) Cystic Thymoma:

- Partially cytsic / partially solid – Cyst with enhancing mural nodules

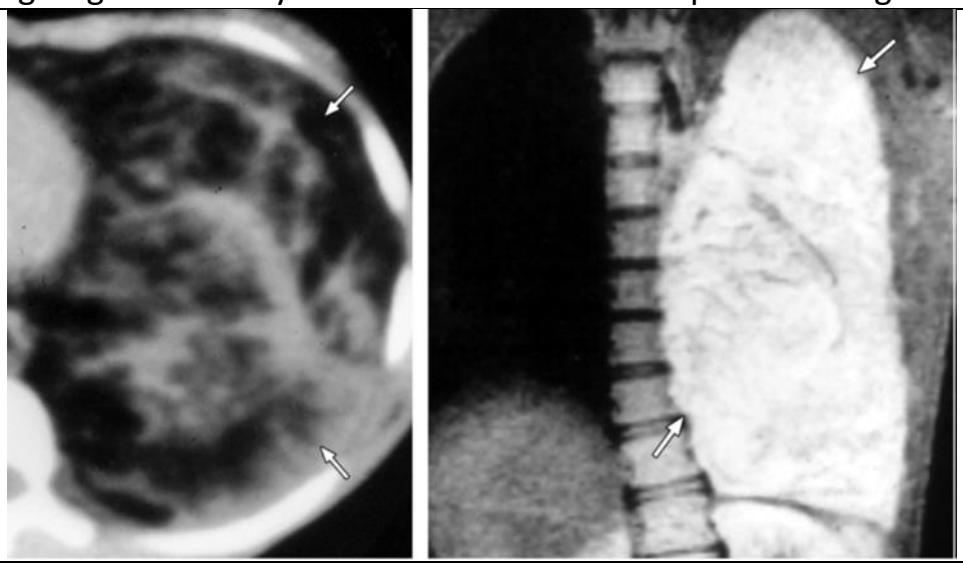


❖ Fatty Lesions

1) Thymolipoma

Rare, benign, well-encapsulated thymic tumors

CT: Fat attenuation + Fibrous septa + Normal thymic tissue
MRI: High signal intensity on T1- and T2 + fibrous septa of low signal.



2) Mediastinal Lipomatosis

- Non-neoplastic accumulation of mature fat in areas of where fat normally occurs.

Associations:

- Obesity.
- Cushing syndrome
- Exogenous steroid therapy

CT:

→ Increased mediastinal fat
→ Increased cardiophrenic fat
→ No soft tissue component
→ No calcification

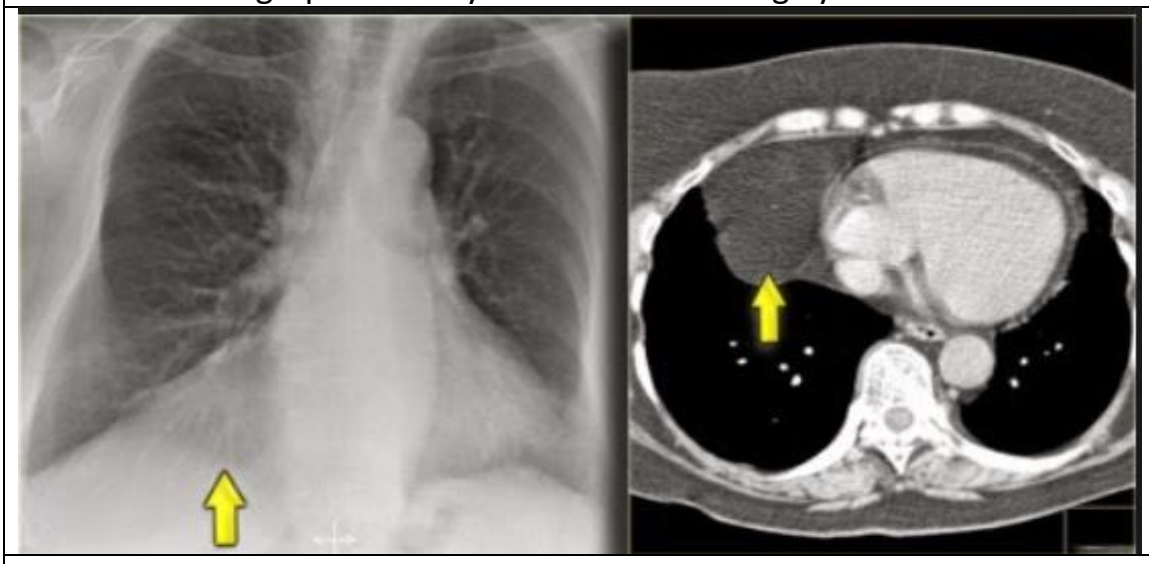
Anterior Mediastinal Mass

B) Precardiac (Inferior Anterior Mediastinum)

Cardiophrenic Angle Lesions

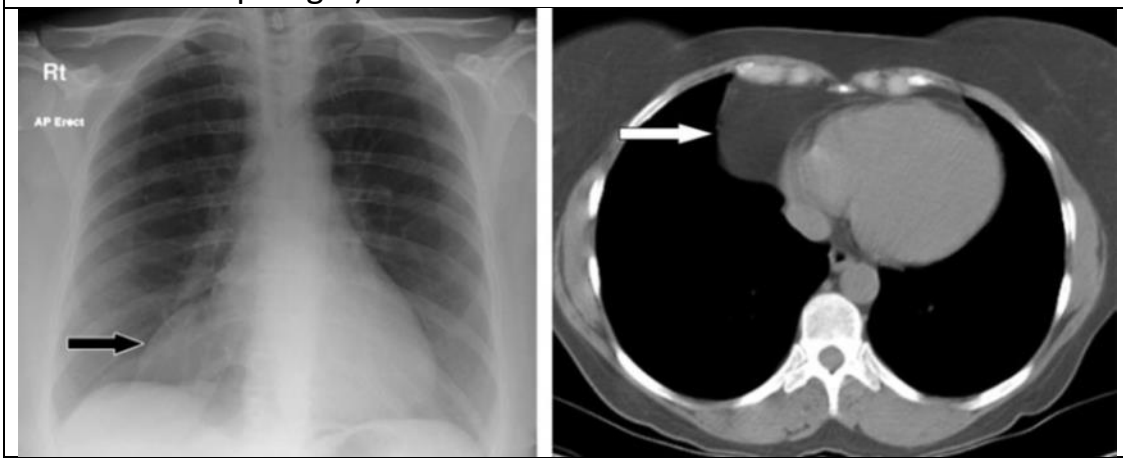
Epicardial Fat Pad

- A prominent epicardial fat pad silhouettes the cardiac border on a frontal radiograph and may simulate cardiomegaly.



Pericardial Cyst

- Benign cystic lesion thought to be congenital.
- Most are located at the right cardiophrenic angle (between the heart and the diaphragm).



Morgagni Hernia

- A diaphragmatic hernia through the foramen of Morgagni
- Usually occurs on the right.
- Containing omental fat and often bowel.
- If bowel gas is absent, a key to diagnosis on CT is the detection of omental vessels in the mass which can be traced into the upper abdomen.

II- Middle Mediastinum

DD:

LNs	Vascular	Cyst
• Lymphoma • TB • Sarcoidosis • Silicosis / CWP • Metastasis • Castleman diseases	• Aortic Aneurysm • Enlarged PA: ○ PAH ○ PS ○ Pulm. Aneurysm ○ Idiopathic pulm. dilatation.	**Foregut duplication cyst:** 1) Bronchogenic cyst 2) Esophageal duplication cysts. 3) Neuroenteric cysts.
Trachea	**Calcification**	
• Tracheal tumors • Tracheomalacia	• Fibrosing mediastinitis	

- Widened paratracheal stripes
- AP window mass
- Obliterated left SCA reflection
- Displaced azygoesophageal recess on the right side
- Pseudo-paraverteberal line on the left side.
- Mass on posterior trachea.
- Lateral Doughnut.

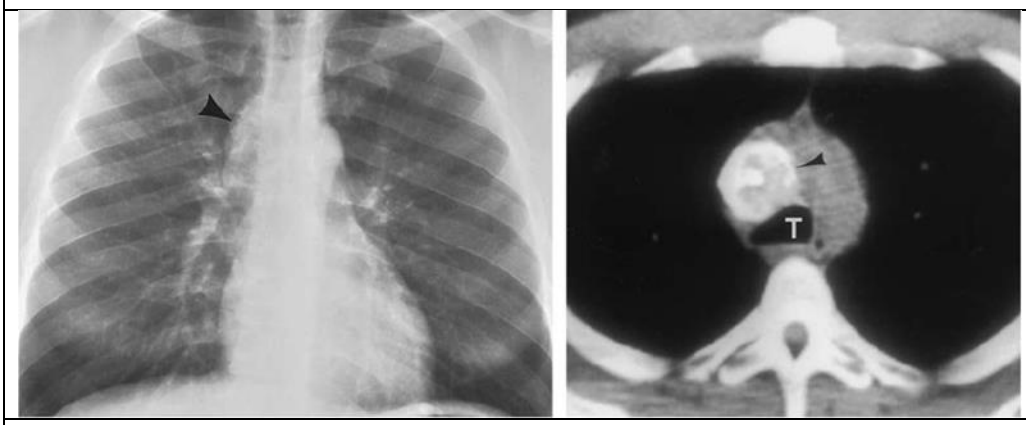
Fibrosing Mediastinitis
Sclerosing Mediastinitis

Idiopathic uncommon benign disorder characterized by proliferation of dense fibrous tissue within the mediastinum

2 Patterns:

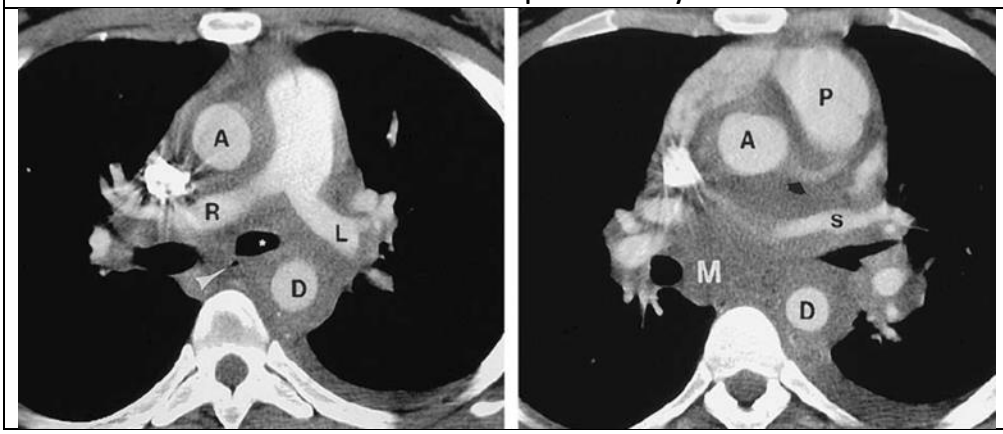
- **Focal type :**

- Localized calcified mass in:
  - Paratracheal region
  - Subcarinal region
  - Hilum.



- **Diffuse type:**

- Diffusely infiltrating, often noncalcified mass that affects multiple mediastinal compartments.
- SVC obstruction => SVC Syndrome
- Encasement or obstruction of pulmonary arteries and veins



III- Posterior Mediastinum

Neurogenic	Vertebral

• Peripheral nerves
  ○ Schwannoma
  ○ Neurfibroma
  ○ Malignant nerve sheath tumor
• Sympathetic ganglia:
  ○ Ganglioneuroma
  ○ Ganglioneuroblastoma
  ○ Neuroblastoma
• Paraganglion cells:
  ○ Paraganglioma
  ○ Pheochromocytoma
	• Congenital:
	  ○ Lateral Meningocele
	• Trauma: Hematoma
	• Infection: Discitis
	  ○ Paraspinal abscess
	• Neoplastic:
	  ○ Metastasis – MM
	  ○ Lymphoma – Leukemia
	  ○ ABC – GCT
	  ○ Osteoblastoma
	  ○ Chordoma
	• Degenerative: Osteophytes

Vascular	**Esophagus**
○ Descending Aortic Aneurysm ○ Azygous continuation of IVC	○ Esophageal tumors ○ Achalasia ○ Hiatus hernia ○ Esophageal diverticulum ○ Foregut duplication cyst.
○ **Bochdalek Hernia**	○ **Extramedullary hematopoiesis**

CXR:

- Disruption of the paraspinal lines
- Disruption of the azygoesophageal recess

❖ Neurogenic Tumors

1) Neuroblastoma

Malignant tumor of primitive neural crest cells
3rd most common pediatric malignancy after leukemia/lymphoma and CNS tumors

Age:

- ✓ **Neuroblastoma < 3y**
- ✓ **Ganglioneuroblastoma 3-10y**
- ✓ **Ganglioneuroma > 10y**

Location:

- Adrenal medulla (35%)
- Extraadrenal retroperitoneum (30-35%)
- Posterior mediastinum (20%)
- Pelvis (2-3%)
- Neck (1-5%)
- Metastatic disease with no primary identified (1%)

Staging:

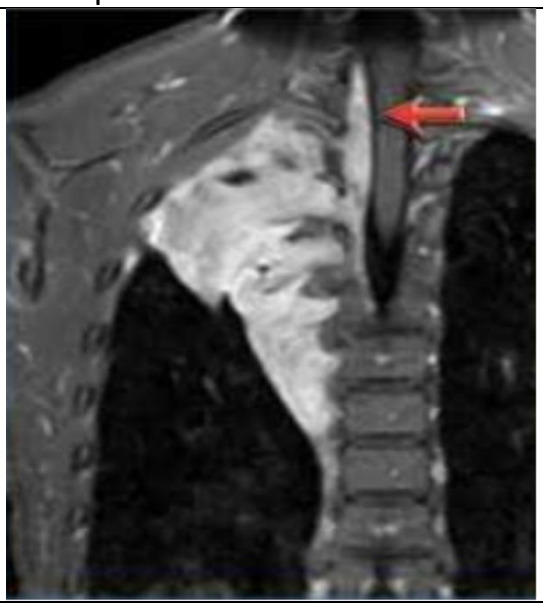
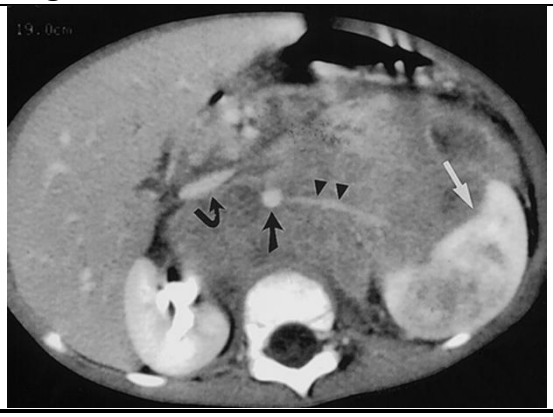
1: Confined to organ of interest (90% survival)
2: Extension beyond organ but not crossing midline (7S% survival)
3: Extension crossing midline (30% survival)
4: Distal metastasis (10% survival)
4S: Age < 1 year, metastatic disease confined to skin, liver, and bone marrow (near 100% survival)

Associations:

Von Recklinghausen disease
Beckwith-Wiedemann syndrome
Hirschsprung disease

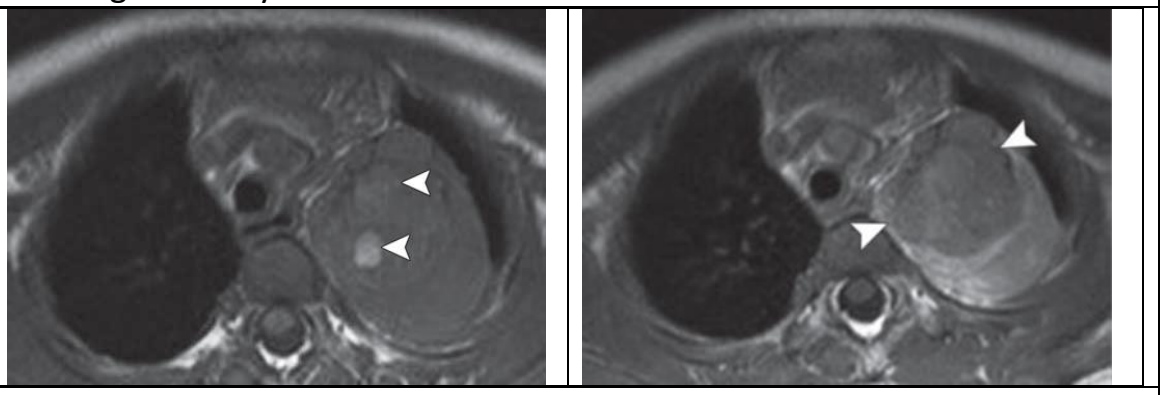
Imaging Findings:

- Calcifications: Common 85%
- Heterogeneous from necrosis and hemorrhage
- Aggressive tumor with tendency to invade adjacent tissues
- Surrounds and engulfs vascular structures
- Spinal canal invasion via neuroforamina
- Metastasizes most commonly to liver and bone

Calcifications	Intraspinal extension
	
Engulf vessels	**Bone metastasis**
	

2) Ganglioneuroblastoma

- ✓ Malignant but less aggressive than neuroblastoma
- ✓ Age = 3-10y



3) Ganglioneuroma

- ✓ Benign
- ✓ Age > 10y

Location:

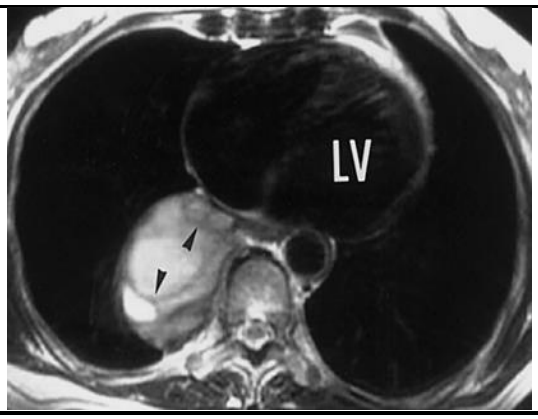
- Posterior mediastinum (41.5%)
- Retroperitoneum (37.5%)
- Adrenal gland (21%)
- Neck (8%).

Imaging findings:

- Well defined lesion
- Calcifications : 42%–60%
- Homogeneous CT density / MR signal
- Mild enhancement
- Rare metastases

T2: "whorled" pattern:

Heterogeneous high T2 signal lesion +
characteristic curvilinear bands of low T2 signal representing bundles of Schwann cells and collagen fibers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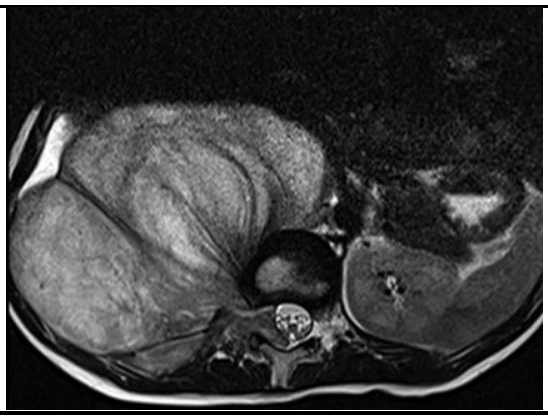
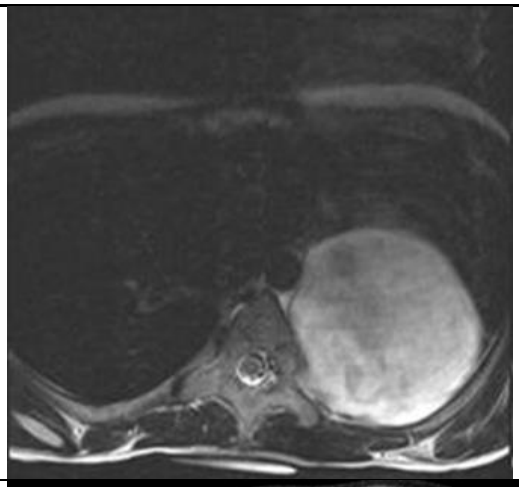
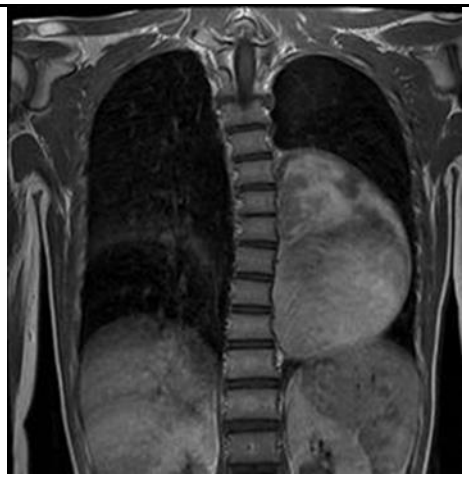
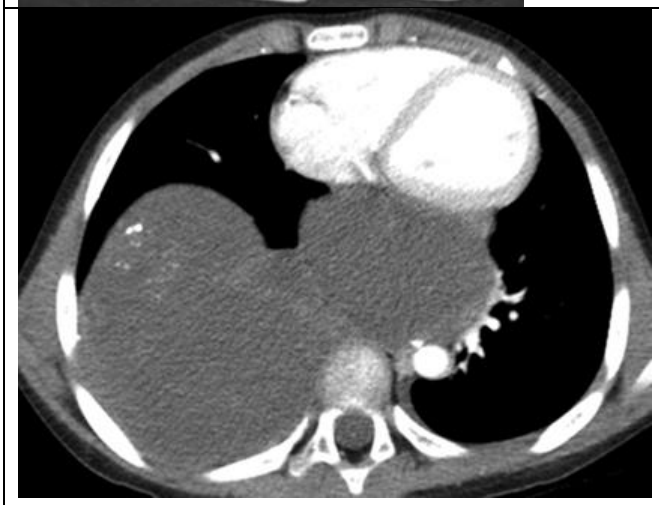








4) Schwannoma

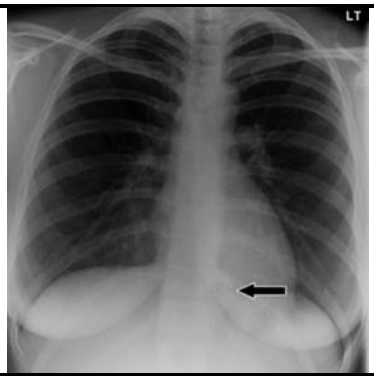
- Most common (50%) mediastinal neurogenic tumor
- Age: 20–30 years
- Sex: F > M
- Solitary
- Multiple schwannomas may be associated with NF-II

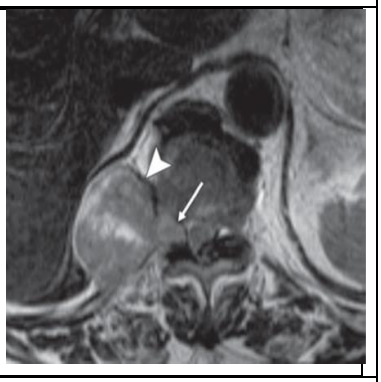
Imaging Findings:

- Posterior mediastinal / paravertebral lesion
- Well defined lesion
- Spherical / oval shaped
- Dumbbell shaped lesion with intra-spinal extension
- Widening of the neural foramen
- Pressure erosions on adjacent bony structures (Rib notching)

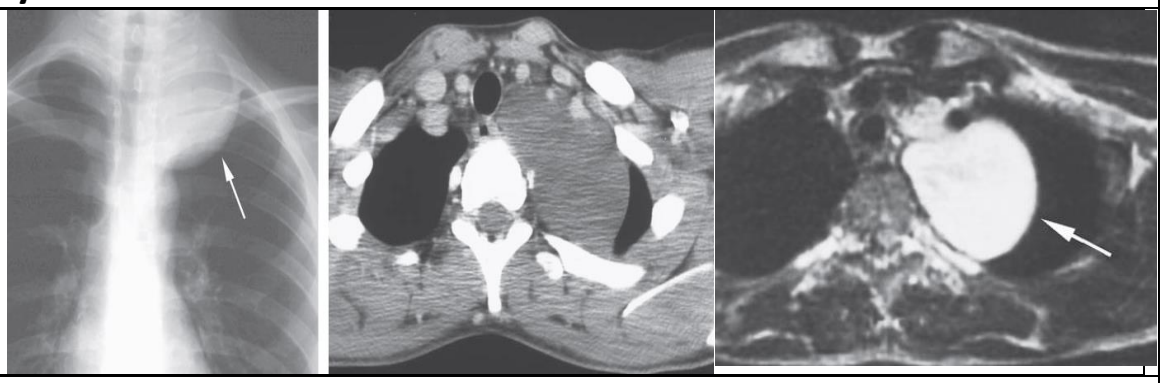
	Antoni A	Antoni B
	Hypercellular	Hypocellular
T2	Low signal	very high signal
T1+C	Strong contrast enhancement	Weak contrast enhancement

T1: Homogenous Low signal
T2: Homogenous high signal ± Cystic areas
+C: Moderate enhancement ± non-enhancing central areas





Cystic Schwannoma



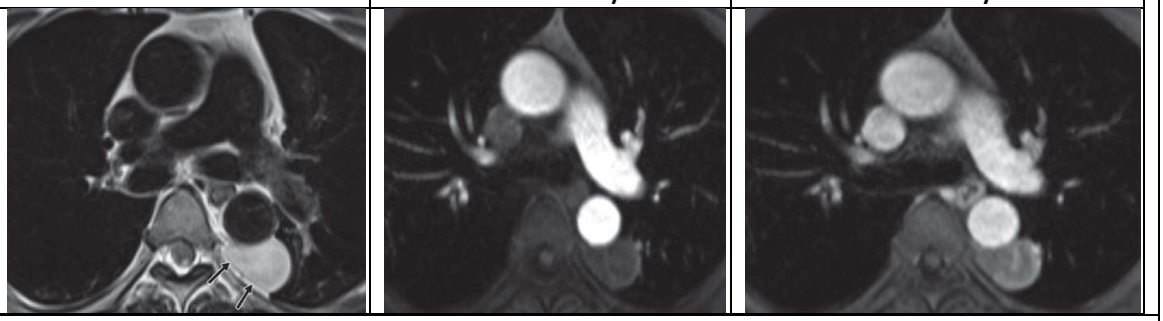
5) Neurofibroma

- Account for 20% of mediastinal neurogenic tumors.
- Age = 20-30 y
- Multiple neurofibromas associated with NF-I

Imaging Findings:

- Well defined lesions
- T1: Low signal
- T2: Target sign: Central high signal and peripheral low signal
- +C: Target sign: Central enhancement.

Axial T2	Axial T1+C Early	Axial T1+C Delayed



6) Malignant Peripheral Nerve Sheath Tumor (MPNST)

Rare spindle cell sarcoma arising from a peripheral nerve or Neurofibroma.
Age = Adult patients 20–50y
associated with NF-I in 25–70% of cases

Commonly involves major nerve trunks, such as:
Sciatic nerve, brachial plexus, sacral plexus, and paraspinal nerves

Imaging Findings:

- Heterogeneous signal due to tumor necrosis and hemorrhage
- Heterogeneous contrast enhancement
- Invasive

7) Paraganglioma

Paraganglioma is a rare neuroendocrine tumor of chromaffin cell origin

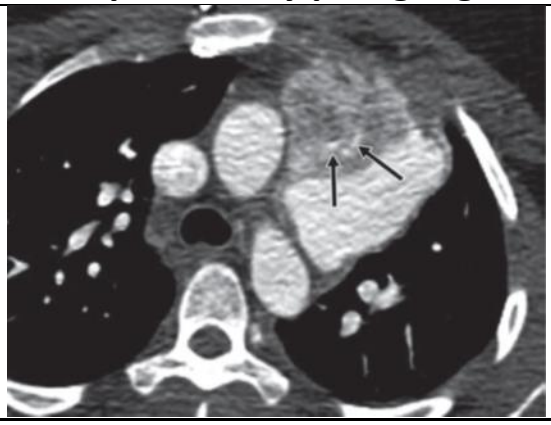
2 locations:

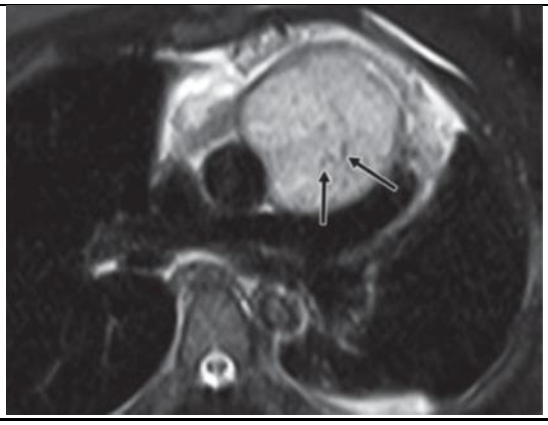
- Aortosympathetic paraganglia = along the sympathetic chain in the posterior mediastinum
- Aortopulmonary paraganglia = along the great vessels

T2: Salt-and-pepper"

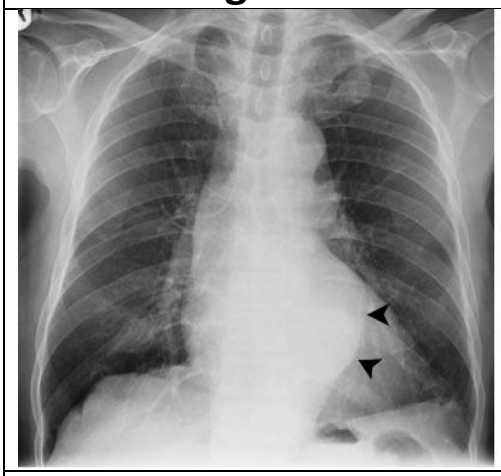
- ✓ The salt component => high-signal-intensity regions, is due to slow flow within tumor vessels or hemorrhage,
- ✓ The pepper component => high flow within tumor vessel
- ✓ Multiple curvilinear and punctate signal voids, which reflect high velocity low in the intratumoral vessels

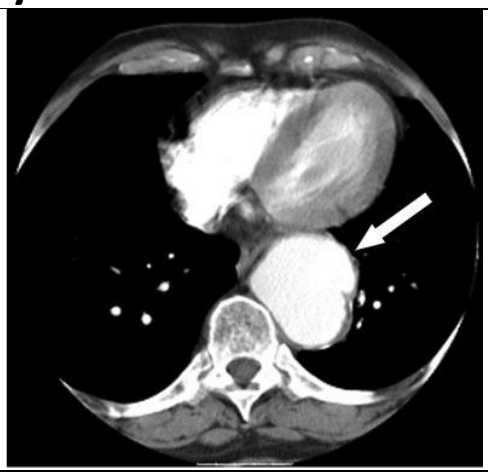
Aortopulmonary paraganglioma



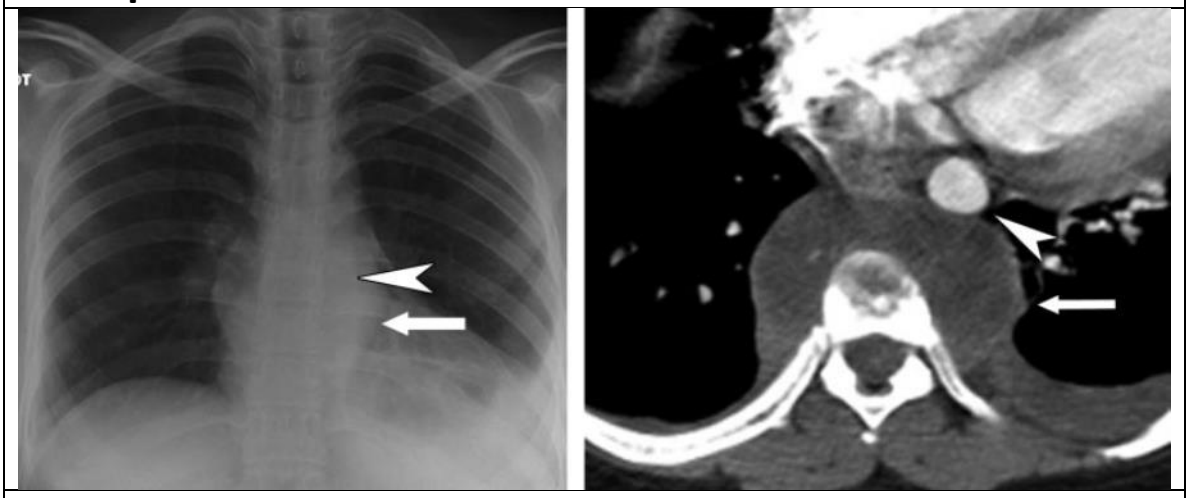


Descending Aortic Aneurysm

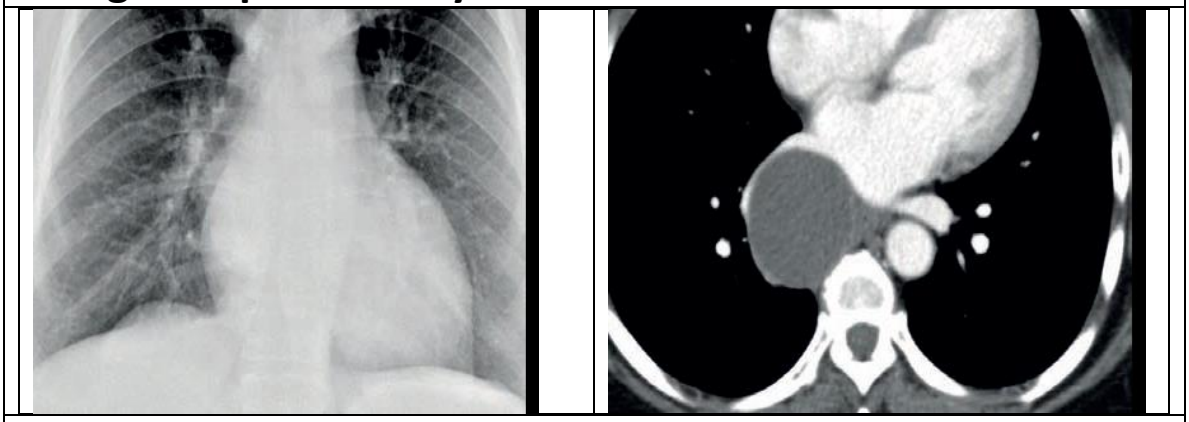




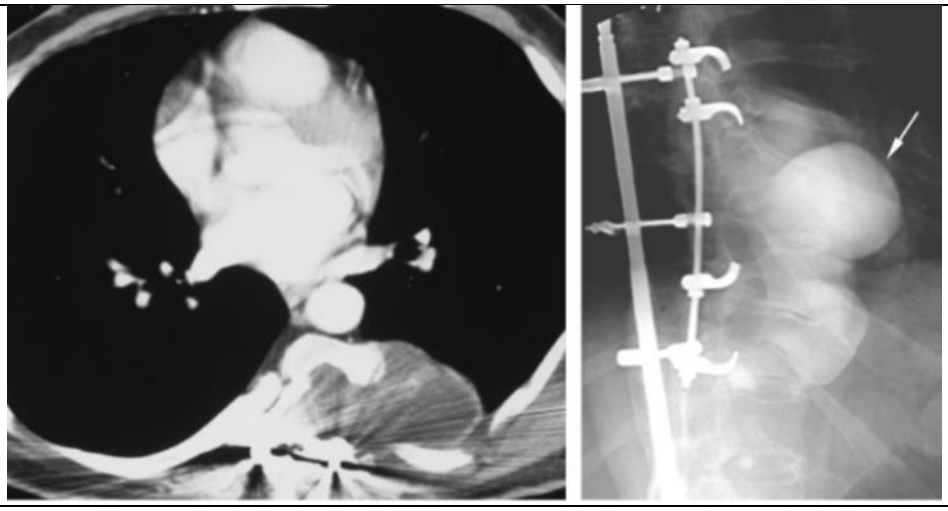
Paraspinal Abscess



Foregut Duplication Cyst



Lateral Thoracic Meningocele In NF-I



IV- Mediastinal Emergency

✓ Acute Mediastinitis

Blood	Air
✓ Spontaneous mediastinal hematoma ✓ Cardiac tamponade	✓ Tension pneumomediastinum ✓ Tension pneumothorax ✓ Tension pneumopericardium
Acute Aortic Syndrome	**Esophageal Emergency**
✓ Aortic Intramural hematoma ✓ Penetrating Ulcer ✓ Aortic Dissection ✓ Aneurysmal leak ✓ Traumatic Aortic Transection	✓ Intramural Hematoma ✓ Mallory Weiss Tear ✓ Boerhaave Syndrome ✓ FB impaction ✓ Fistula ✓ Esophagitis

Acute Mediastinitis

Acute mediastinitis is a life-threatening condition with high mortality and morbidity.

Causative organism:

- Staphylococcus aureus is the most common cause

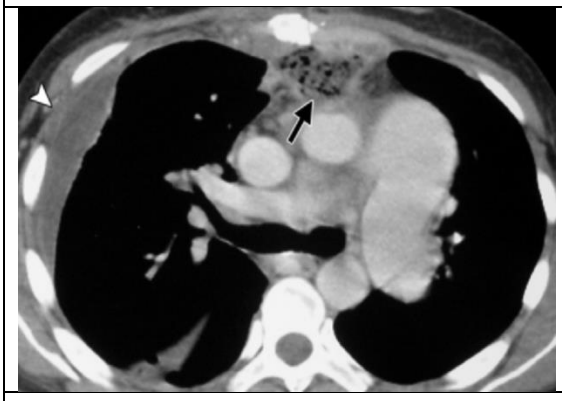
Predisposing Factors:

- Postoperative
- Secondary to esophageal perforation
- Spread of osteomyelitis from adjacent bone
- Direct extension of head and neck infection
- Hematogenous spread of infection

CT findings:

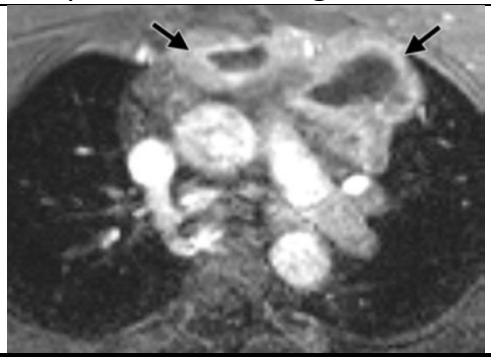
- Increased attenuation of mediastinal fat
- Free gas bubbles in the mediastinum
- Localized fluid collections
- Enlarged lymph nodes
- Pleural effusions, and empyema

Air and fluid collections (arrow) in the anterior mediastinum



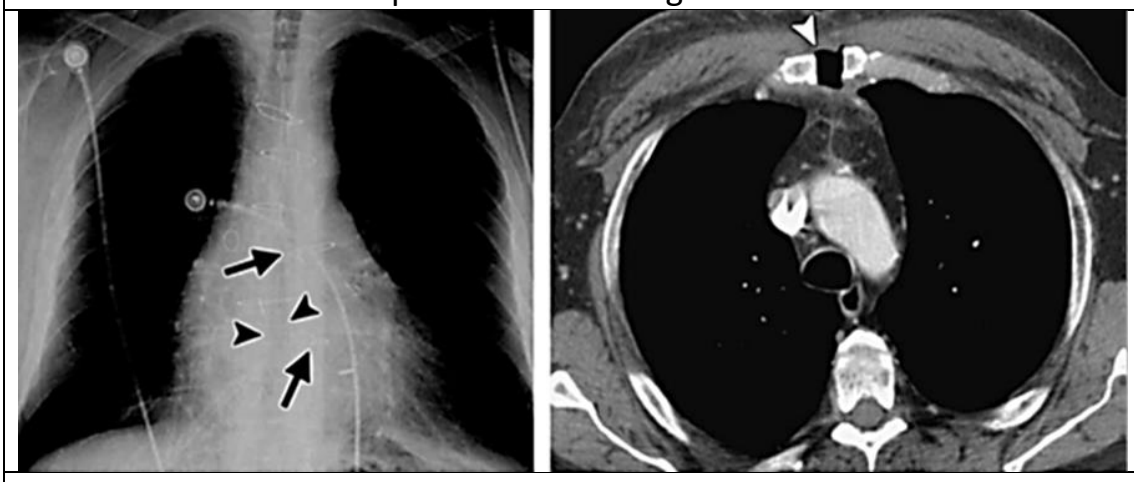
Septic arthritis of the left sternoclavicular joint.

Peripheral enhancing mediastinal fluid collection



Sternal Dehiscence:

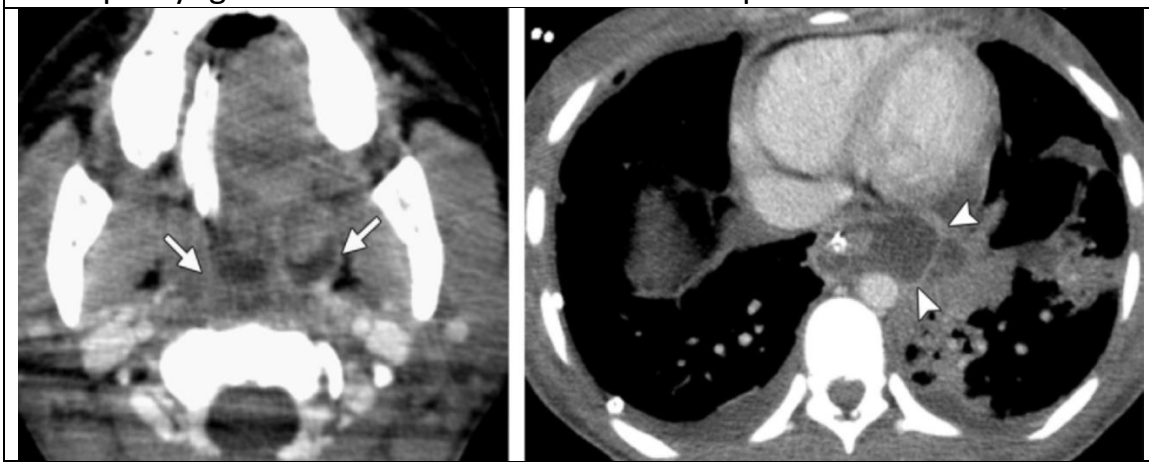
- Displacement, rotation, and fracture of the sternal wires.
- Midsternal lucent stripe on axial CT images > 3 mm



3 Potential pathways for spread of infection from the neck to the mediastinum:

1) Pretracheal route to the anterior mediastinum
2) Lateral pharyngeal route to the middle mediastinum
3) Retropharyngeal-retrovisceral route to the posterior mediastinum.

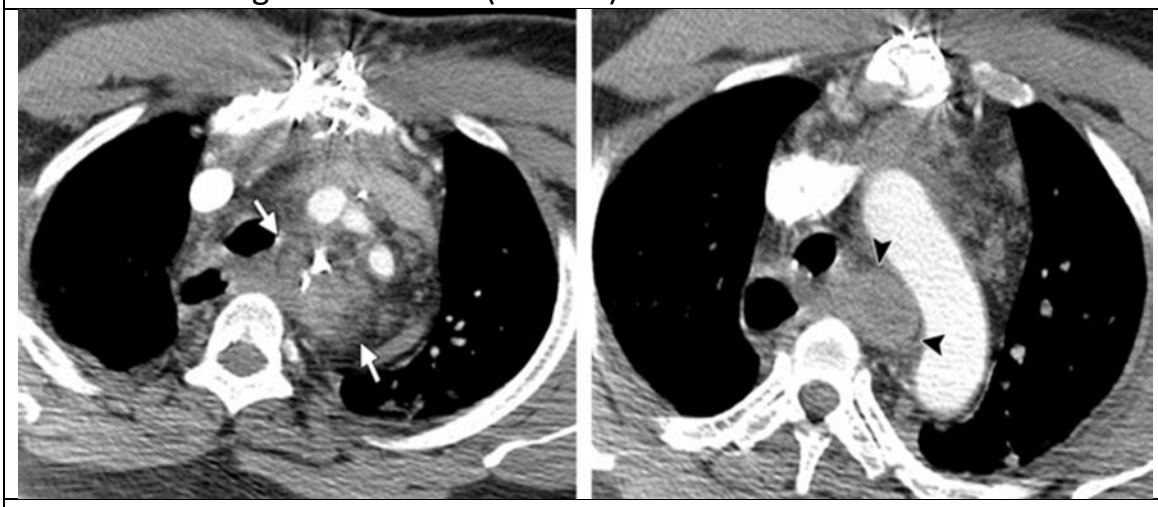
Retropharyngeal Abscess => Fluid collections in posterior mediastinum



Spontaneous Mediastinal Hematoma

a) Bleeding disorders or who are undergoing anticoagulation therapy;
b) Sudden increase in intrathoracic pressure due to coughing, vomiting.
c) Sudden rise in systemic blood pressure due to malignant hypertension
d) Secondary to hemorrhage into a mediastinal organ or mass with no underlying bleeding disorder

NCCT: a mediastinal hematoma appears as a soft-tissue–attenuation mass with areas of high attenuation (>60 HU).



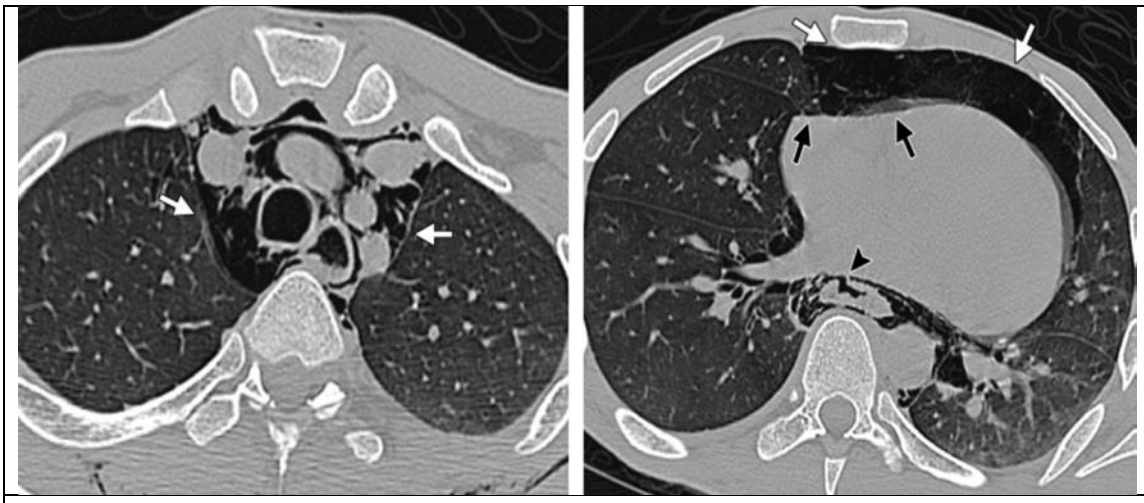
Tension Pneumomediastinum

Common causes:

- Blunt or penetrating trauma
- Esophageal perforation
- Recent interventions in the esophageal or tracheobronchial tree
- Pulmonary infections, gas-forming infections in the mediastinum
- Cocaine inhalation
- Extension of air from a pneumothorax

CT Findings:

- Substantial mediastinal free air
- Flattening of the anterior cardiac contour
- Compression of the right atrium
- Uplifting of the heart off of the diaphragm
- Distention of IVC.
- Compression of the mediastinal vessels
- Flattening of the main bronchi



Tension Pneumopericardium

Trauma (Most common cause):

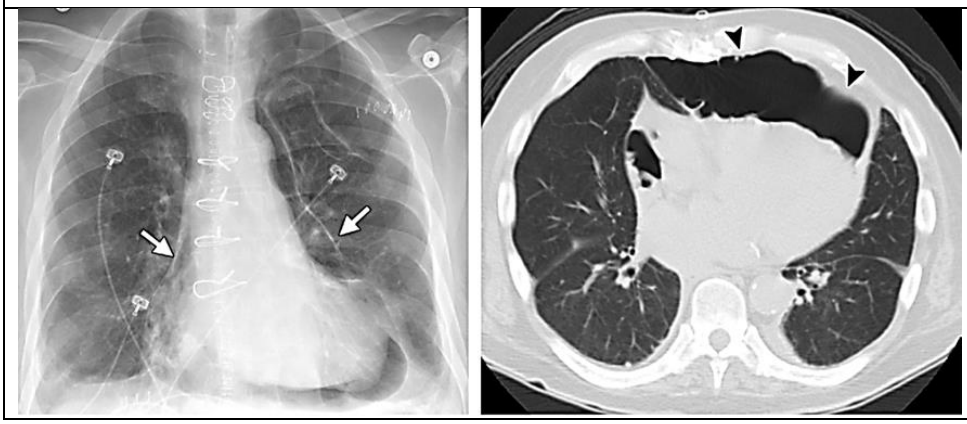
- Blunt or penetrating
- Iatrogenic (eg, occurring during or after cardiac surgery)
- Barotrauma secondary to positive pressure ventilation.

Nontraumatic causes

- Pericarditis with gas-forming organisms,
- Direct extension of an inflammatory process from adjacent structures,
- Fistulous communication with air-containing structures (eg, the stomach, esophagus, airways, and lungs),
- Extension of a pneumomediastinum into the pericardial cavity

CT Findings:

- "halo" sign: continuous rim of air outlining the left and right borders of the heart.
- "small heart" sign: sudden decrease in the size of the cardiac silhouette.
- CT: substantial amounts of air in the pericardial cavity
- Compression and displacement of the heart
- Collapse of the heart chambers,
- Flattening of the anterior border of the heart,
- Distention of the inferior vena cava



Thoracic Trauma

Mechanism of injury:

→ Blunt Trauma: e.g.: Motor vehicle accident
→ Penetrating trauma: e.g.: Stab wound
→ Iatrogenic trauma

A- Thoracic Cage Injuries:

1) Large, open, chest wall defect.
2) Chest wall hematoma
3) Rib fractures.
4) Sternal fractures.
5) Spinal fractures.
6) Clavicle fracture

B- Parenchymal Injuries:

1) Pulmonary contusion
2) Pulmonary laceration
3) Torsion of lung
4) ARDS
5) Fat embolism
6) Atelectasis

C- Pleural Injuries: (Discussed in Pleura Chpater)

1) Pneumothorax
2) Hemothorax

D- Diaphragm Injury:

1) Diaphragmatic tear

E- Mediastinal Injury

1) Mediastinal emphysema
2) Mediastinal hematoma
3) Esophageal injury
4) Vascular Injury
5) Tracheobronchial Injury

F- Cardiac Injury

1) Cardiac Tamponade
2) Pericardial tear
3) Myocardial contusion
4) Myocardial rupture
5) Ventricular aneurysm
6) Septal defect
7) Valvular dysfunction
8) Coronary artery Injury

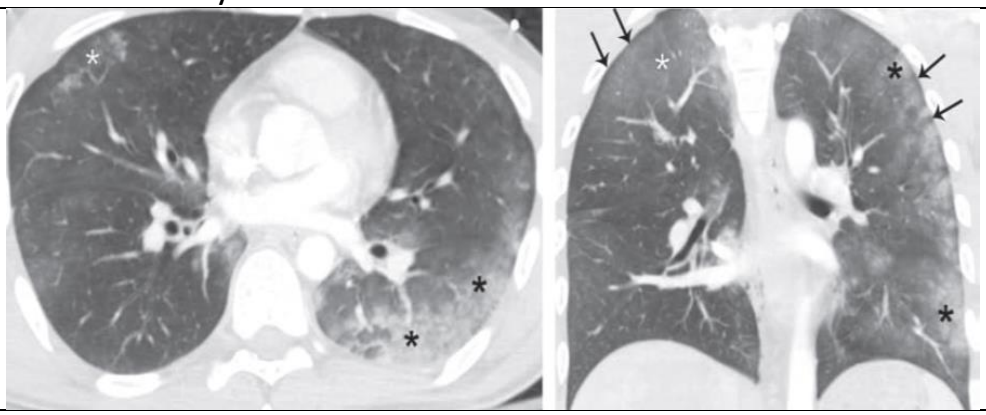
Parenchymal Lung Injuries

1) Pulmonary Contusion

- The most common lung injury from blunt chest trauma
- It represents traumatic injury to the alveoli with alveolar hemorrhage,
- but without significant alveolar disruption

CT:

- Pattern: Ill defined patchy airspace opacities / consolidations
- Distribution: Peripheral with subpleural sparring
- Appear: 6-12hrs after trauma
- Resolve: 7-10 days

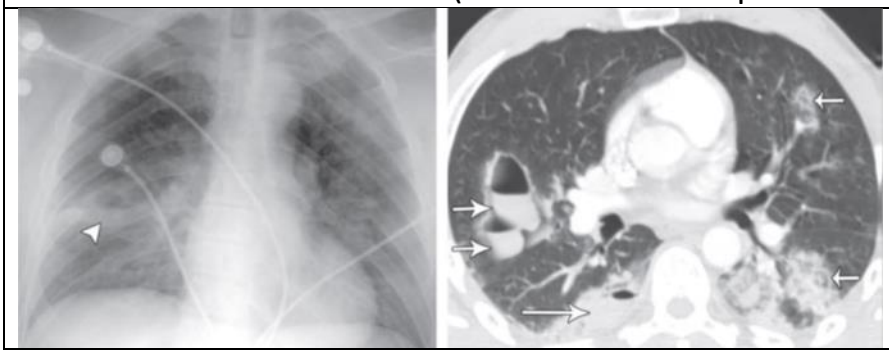


2) Pulmonary Laceration

- Disruption (tear, laceration) of the lung parenchyma, resulting in a cavity in the lung.

CT:

- Round or oval cavity filled with:
    - Air (traumatic pneumatocele)
    - Blood (traumatic hematocele or pulmonary hematoma)
    - Both air and blood (traumatic hematopneumatocele)

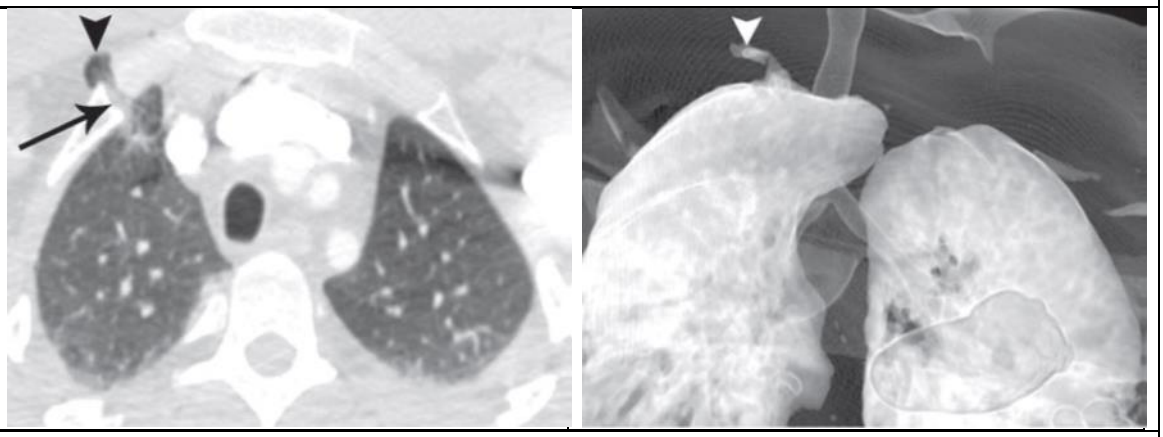


4 Types of Lung Laceration:

Type	Location	Pneumothorax	Mechanism
1	Mid lung	Variable	Compression rupture injury = Shear between parenchyma and tracheobronchial tree
2	Paraspinal	Uncommon	Compression shear injury = Shear due to sudden herniation of lower lobe parenchyma in the front of vertebral column
3	Subpleural	Usual	Rib penetration tear = Puncture by displaced rib fracture
4	Subpleural	Usual	Adhesion tear = Shear at the site of the transpleural adhesion

3) Traumatic Lung Herniation

Pleura covered part of the lung extrudes through a traumatic defect in the chest wall.



4) Fat Embolism

- Lipid emboli from bone marrow enter pulmonary and systemic circulation.
- Suspected in patients with sudden onset of dysopnea and multiple fracture.
- Pattern: Interstitial and alveolar hemorrhagic edema opacities
- Onset: delayed (48hrs ahfter the incident)
- Clear: 3-7 days

Airway Injuries

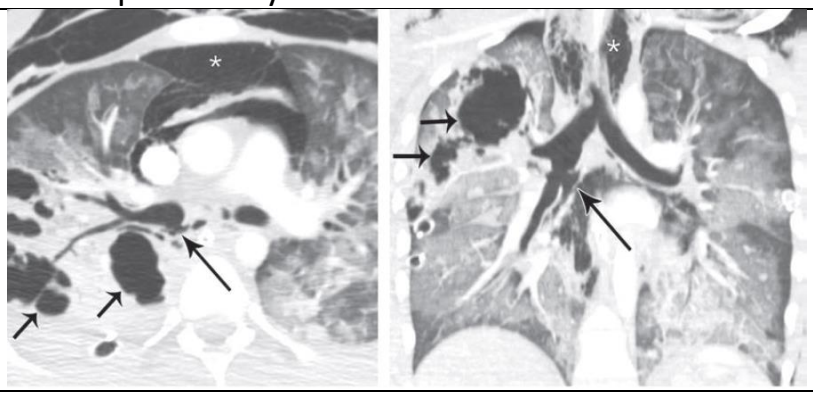
Rare. High mortality (30%)

1) Bronchial Laceration:

- Bronchial lacerations are more common than tracheal lacerations
- Typically parallel to the cartilage rings of the bronchi
- RT main stem > LT distal bronchus
- 75% occur within 2cm of tracheal carina

CT: Pneumomediastinum – Pneumothorax

Tear (long arrow) in the posterior wall of the right intermediate bronchus with an air leak into the mediastinum (*) and a small right pneumothorax.
Multiple right pulmonary lacerations
Extensive bilateral pulmonary contusions



<u>Complete bronchial transection:</u>

"Fallen lung" sign = The lung on the side of the bronchial injury may fall posterolaterally away from the hilum

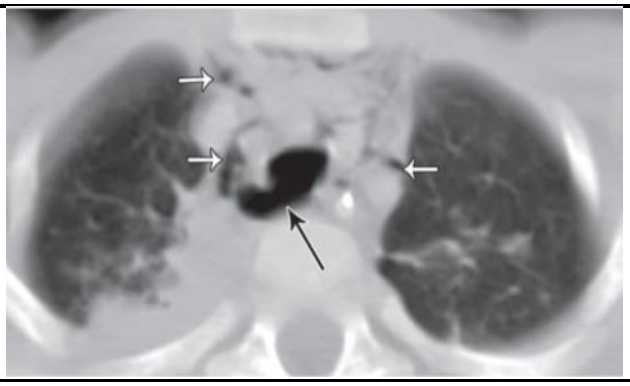
2) Tracheal Lacerations

Vertical and longitudinal and located at the junction of the cartilaginous and membranous portions of the trachea.

CT:

- **Cervical subcutaneous emphysema**
- **Pneumomediastinum.**
- **Pneumothorax**

In intubated patients, overdistention or herniation of an endotracheal balloon



Chest Wall Injury

1) Simple Rib Fracture

Fractures of 1st three ribs

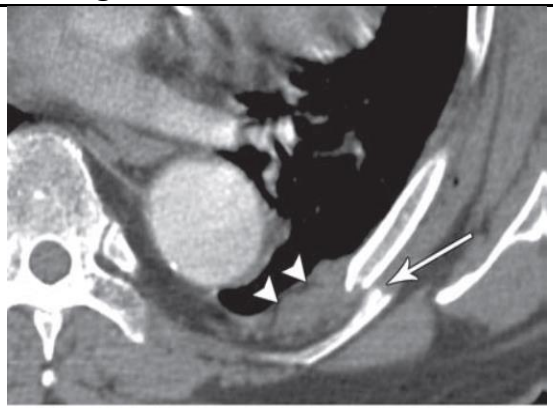
- High-energy trauma because these ribs are well protected by the scapulae, clavicles, and musculature.
- Associated with brachial plexus injury or subclavian vascular injuries.

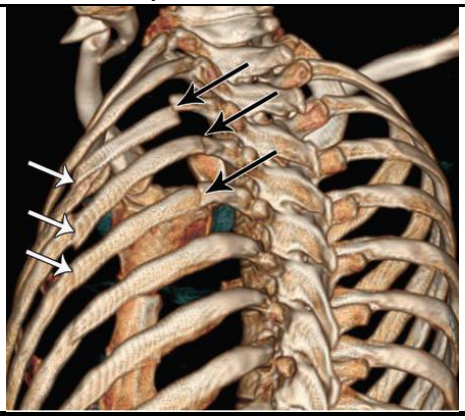
Fractures of lower three ribs

- Associated with liver, spleen, and kidney injuries

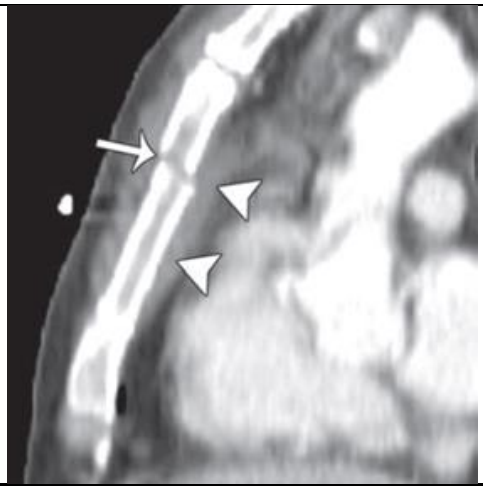
2) Flail Chest

2-3 contiguous rib fractures at 2 - 3 planes
Flail segment => Paradoxical movement with respiration

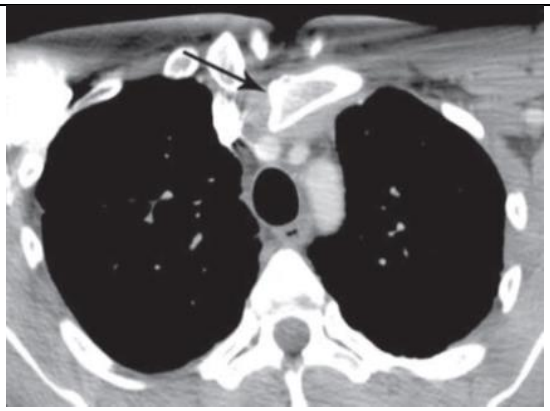




3) Sternal Fracture



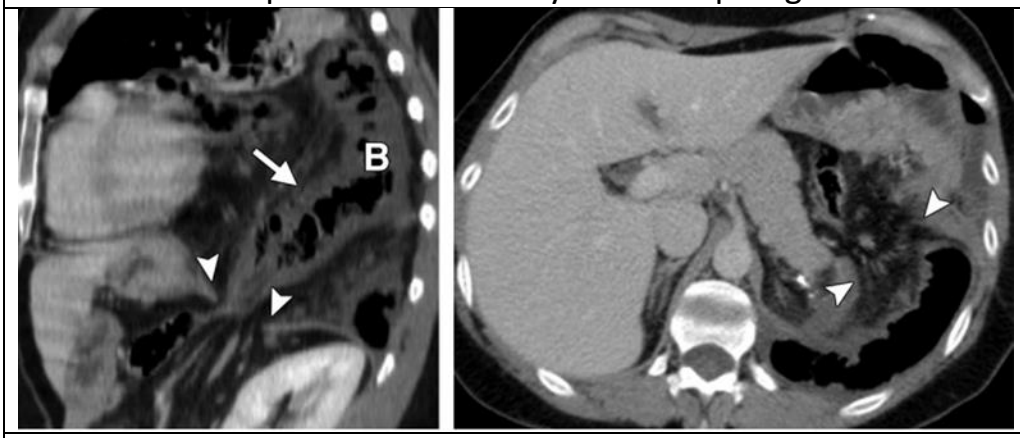
4) Sternocalvicular Dislocation



Diaphragmatic Injury

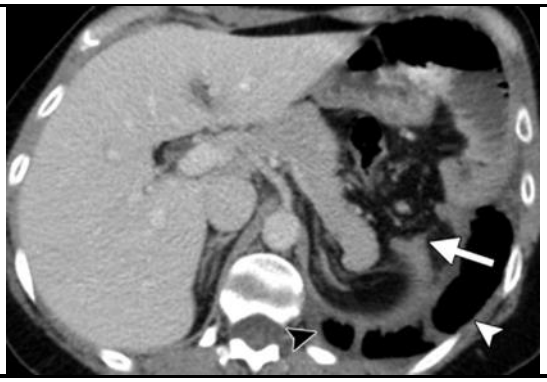
1) Segmental Diaphragmatic Defect:

A focal and abrupt loss of continuity in the diaphragm



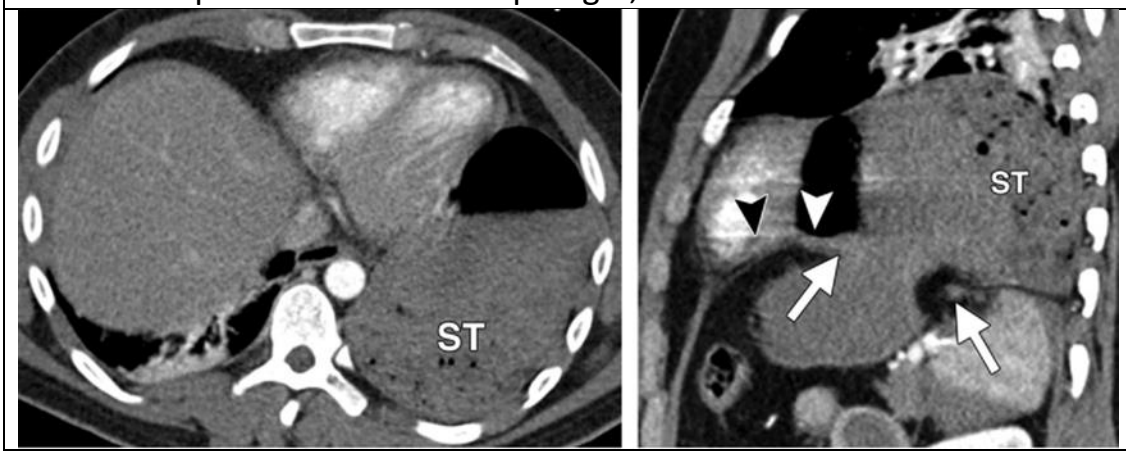
2) Dangling Diaphragm:

The free edge of the torn diaphragm, curls inward from its normal course, forming a comma-shaped or curvilinear structure with soft-tissue attenuation at a near right angle with the chest wall.



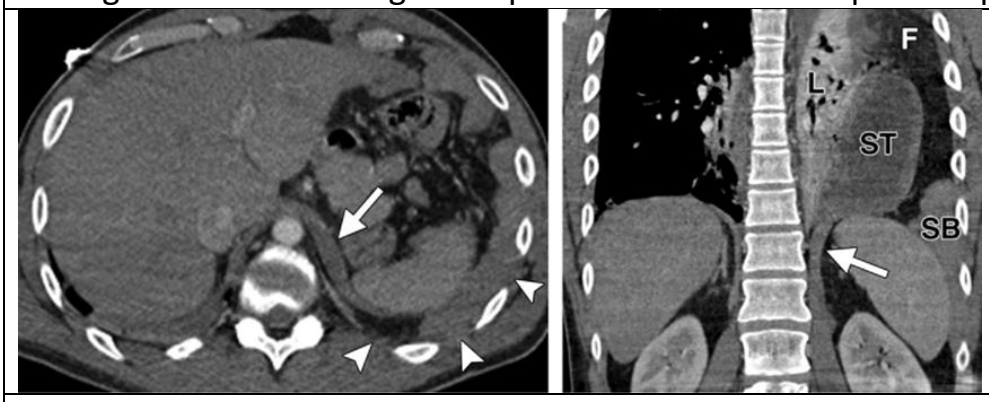
3) Absent Diaphragm sign:

Absence of part or all of hemidiaphragm, without demonstration of tear



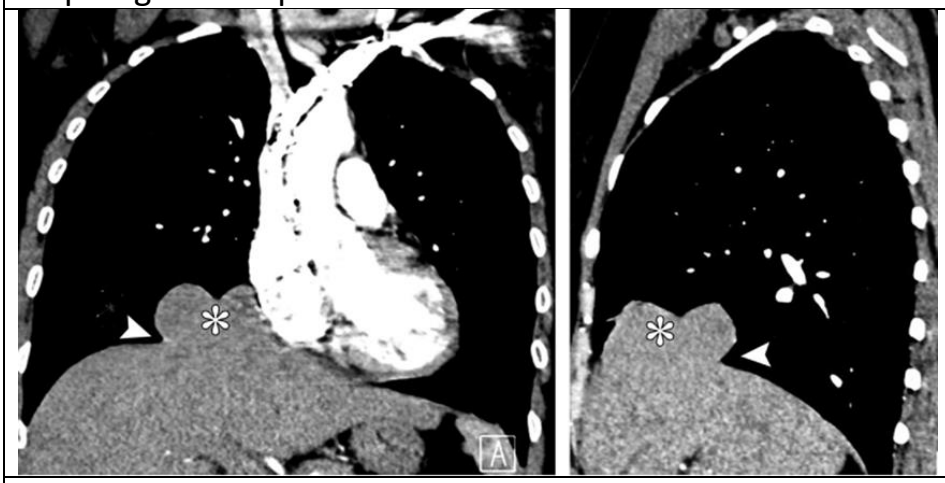
4) Herniation through a Defect

Passage of abdominal organs or peritoneal fat into the pleural space



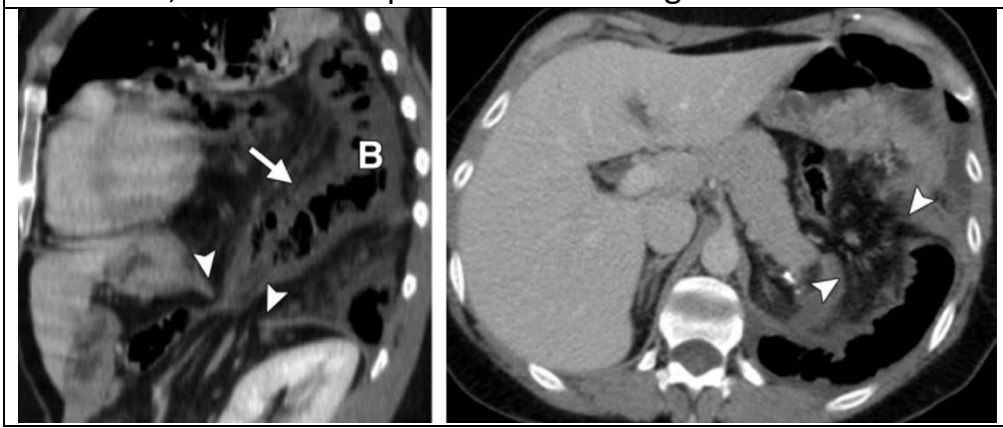
5) Collar Sign

A waist like constriction of the herniated structure at the site of the diaphragmatic rupture



6) Dependent Viscera Sign

direct contact between the herniated abdominal organs and the posterior chest wall, without interposition of the lung

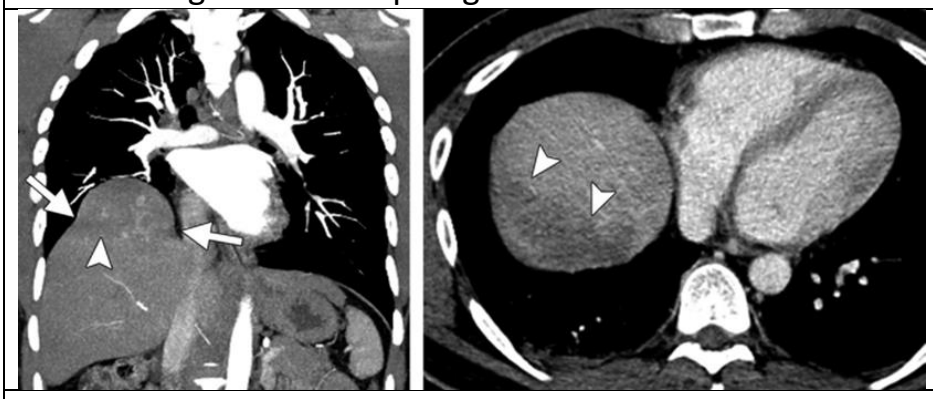


7) The Hump Sign

The shape of the herniated liver located above the level of the diaphragm

8) The Band Sign

A linear area of hypoattenuation that transects the herniated liver between the torn edges of the diaphragm

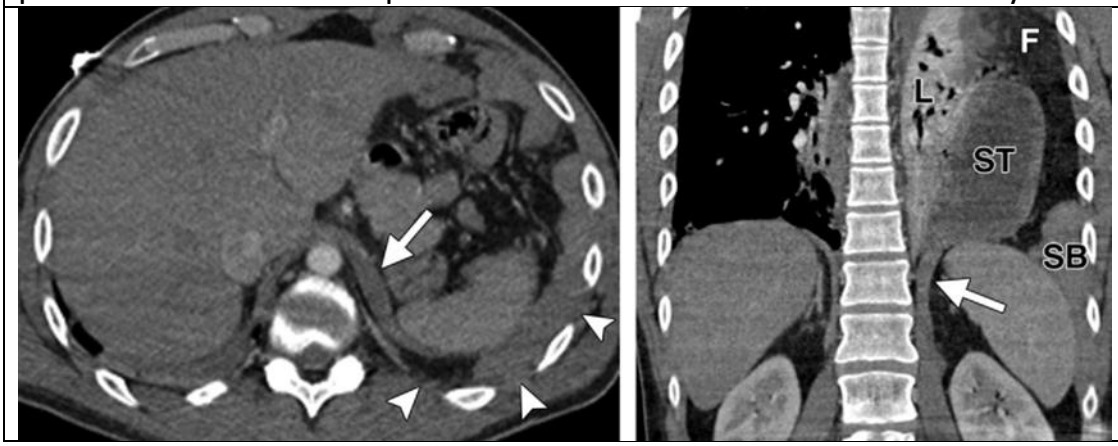


9) Sinus Cutoff

The herniated abdominal contents prevent the expected layering of pleural effusion

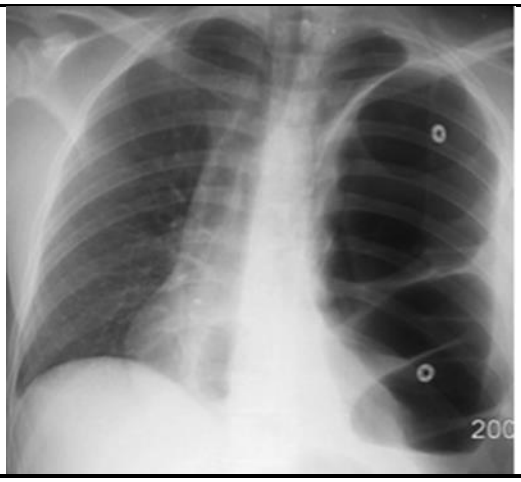
10) Abdominal Content Peripheral to the Diaphragm or Lung

Abdominal organs or fatty tissue seen peripheral to the diaphragm or posterior to the crura represent herniation into the thoracic cavity



11) Elevated Abdominal Organs

Produced by the displacement of abdominal structures cephalad, above the level of the contralateral hemidiaphragm on supine images



Indirect CT Signs Related to Loss of the Border between the Thorax and Abdomen

12	Abdominal fluid abutting a thoracic structure
13	Abdominal viscera abutting thoracic fluid or a thoracic organ
14	Pneumothorax and pneumoperitoneum
15	Hemothorax and hemoperitoneum

CT Signs of Uncertain or Controversial Origin

16	Thickening of the diaphragm	Curled diaphragm sign
17	Diaphragmatic or peridiaphragmatic contrast medium extravasation	...
18	Hypoenhanced diaphragm	Hypoattenuated diaphragm
19	Fractured rib	Presumed laceration of diaphragm by rib, trajectory sign

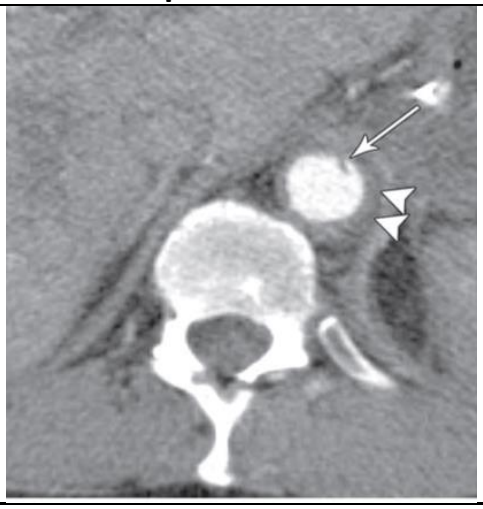
Acute Traumatic Aortic Injury

Location:

- Aortic isthmus: 95%: between left subclavian artery and ligamenturn arteriosum
- Aortic Root: Proximal ascending aorta
- Aortic Hiatus: Descending aorta.
- Associated great vessel injury in 5%-10

5 Direct CTA findings

- ✓ **Aortic intimal flap**
- ✓ **Pseudoaneurysm**
- ✓ **Aortic thrombus**
- ✓ **Adventitial hematoma**
- ✓ **Aortic transection**

Aortic ismuth **Ao psuedoaneurysm**	**Aortic hiatus** **Intimal flap + Periaortic hematoma**
	

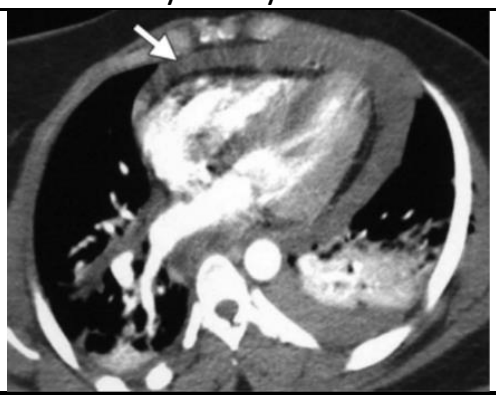
Indirect findings of Mediastinal hematoma:

- Widened mediastinum
- Right paratracheal widening
- Loss of aortic contour
- Left apical cap
- Rightward displacement of nasogastric tube
- Downward displacement of left bronchus
- Fracture of first and second ribs
- Hemothorax

Cardiac Trauma

1) Traumatic Heamopericardium = Cardiac Tamponade

- Cardiac rupture (ventricular or atrial) (high mortality rate (76%)
- Aortic root injury
- Myocardial contusion
- Coronary artery laceration



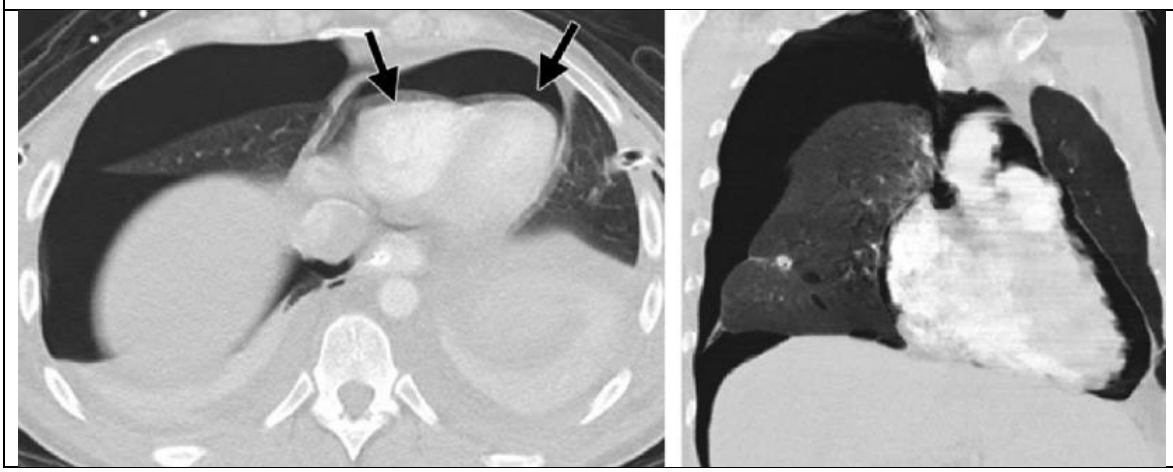
<u>CT findings:</u>

- High-attenuation pericardial effusion
- Distention of IVC, neck and renal veins
- Periportal low-attenuation fluid

2) Traumatic Pericardial Rupture

- Tearing may involve either
  - Pleuropericardium (left, right, or superior)
  - Diaphragmatic pericardium.

- **Pneumopericardium**
- Focal pericardial dimpling and discontinuity
- **Lung interposing** between aorta and pulmonary artery, heart and diaphragm, or right atrium and right ventricular outflow tract
- The “collar” sign: caused by constriction of the cardiac contour by the pericardial opening
- A deformed ventricular silhouette

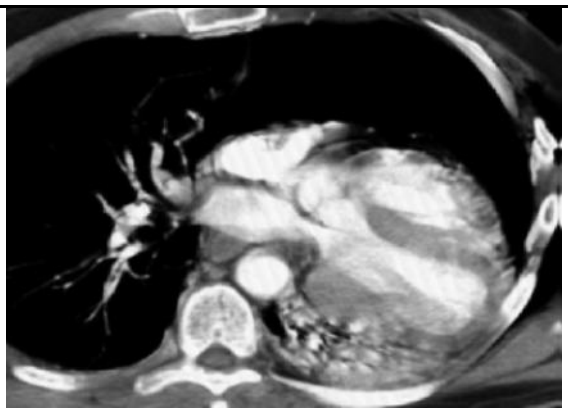


3) Cardiac Herniation

- is a serious complication of pericardial rupture with high mortality rates,
- Partial or complete trans-defect cardiac herniation.

- **<u>Cardiac luxation = Cardiac herniation + Volvulus</u>**
  → Superior vena cava obstruction
  → Strangulation of the heart

- Cardiac displacement into hemithorax cavity
- Empty air-filled pericardial sac
- Deviation of myocardial septum to the left
- Left atrium and ventricle entrapment
- Cardiac herniation through a pericardial defect,
- Pneumopericardium, especially when associated with pneumothorax

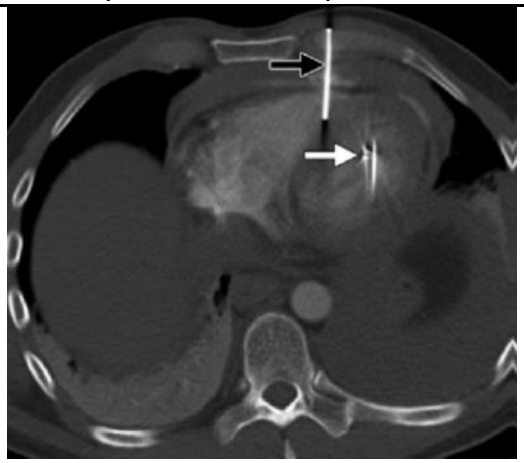


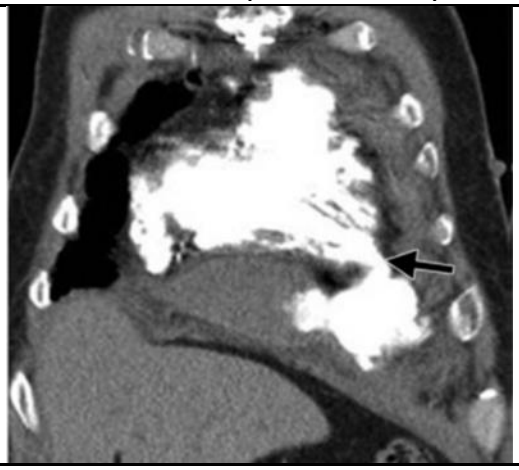
4) Myocardial Contusion

Rt > Lt due to anterior postion and thinner wall
Regional myocardial dysfunction and perfusion defects that occur in the absence of coronary artery disease

5) Myocardial Rupture

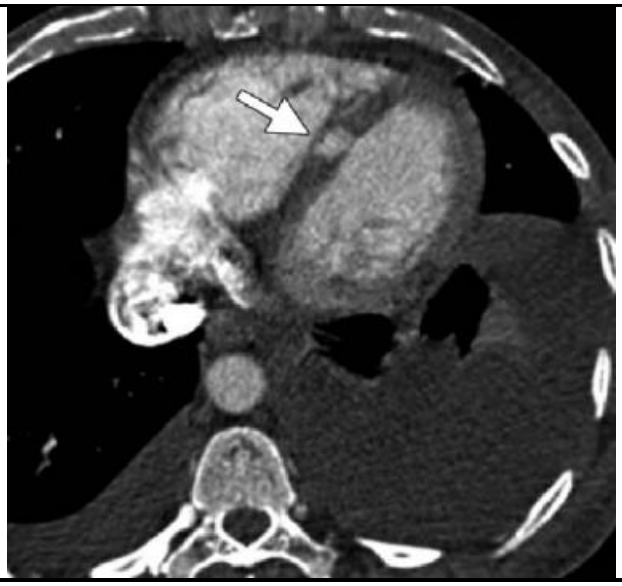
Rt > Lt due to anterior postion and thinner wall
Lethal
CTA: Myocardial disruption + Extravasation of CM into pericardial space





6) Traumatic Ventricular Septal Defect

- Occur in the muscular portion of the interventricular septum, near the cardiac apex
-
- Result from AP compression of the heart between the sternum and the thoracic spine



7) Traumatic Valvular Dysfunction

- Valvular dysfunction due to trauma is relatively uncommon
- Should be considered if pulmonary edema develops after chest trauma.
- The aortic valve is most commonly injured
- Followed by the mitral and tricuspid valves

Mechanism of injury:

- Tear or avulsion from the annulus
- Rupture of the papillary muscles
- Rupture of the chordae tendineae
- Leaflet tear

8) Coronary Artery Injury

- Injury to the coronary arteries is **rare**
- Left anterior descending coronary artery is most common site of injury
- Right coronary artery injury can also occur, since it is located right behind the sternum.
- Left circumflex artery is rarely injured

Esophageal Trauma

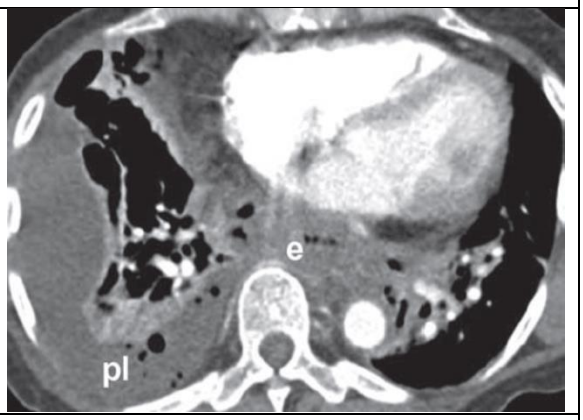
1) Esophageal Perforation:

- **Trauma**
- **Neoplastic (eg, esophageal or lung cancers)**
- **Spontaneous = Boerhaave's Syndrome:**
  - Complete transmural laceration of the esophagus that results from violent straining or vomiting

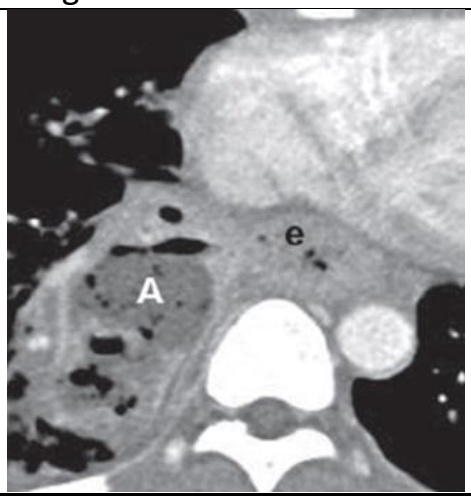
Radiological Findings:

- Focal esophageal wall thickening
- Periesophageal fluid collections
- Free mediastinal air
- Oral contrast extravasation into the mediastinum and pleural space

Oral Contrast extravasation

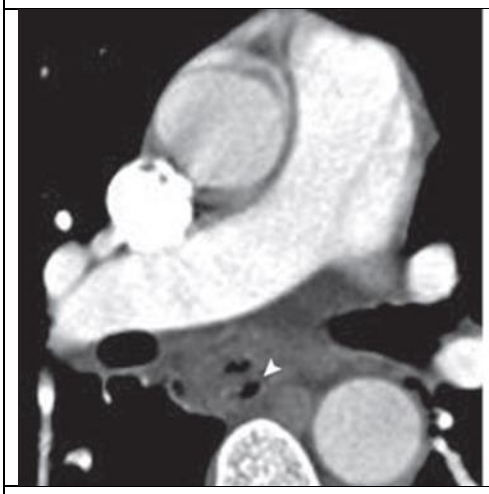


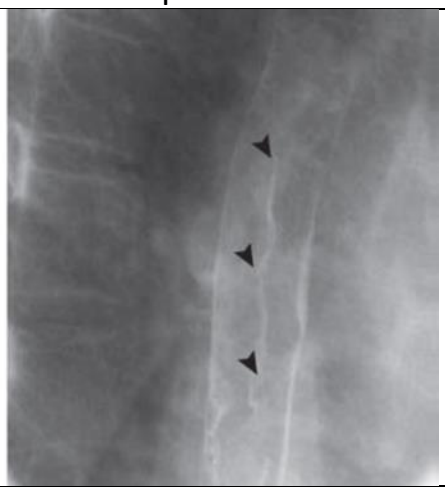
Lung abscess



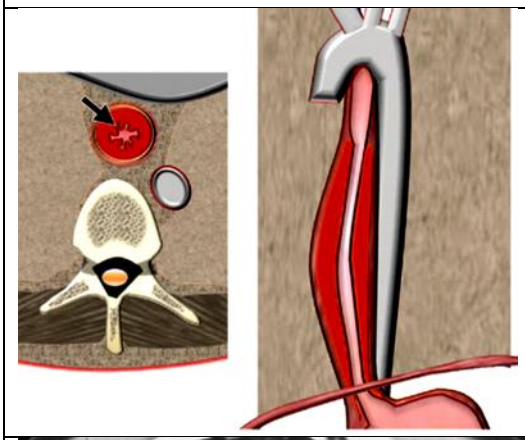
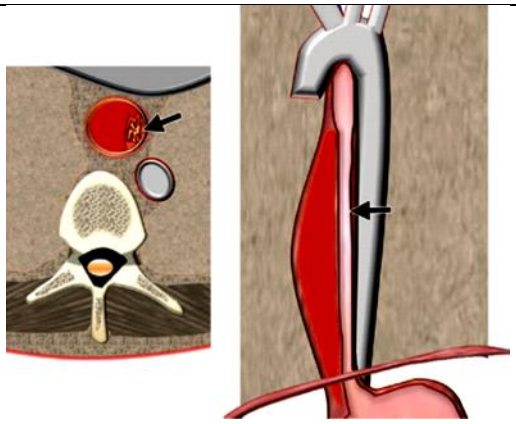
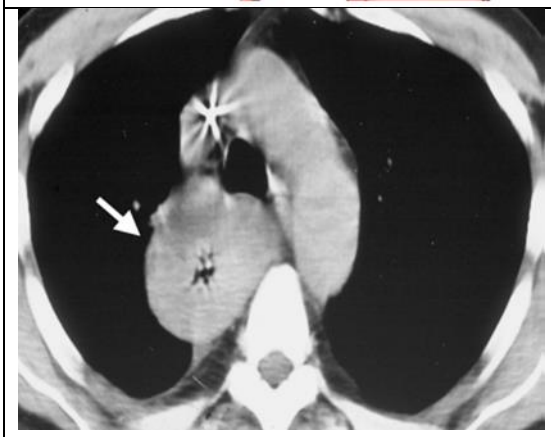
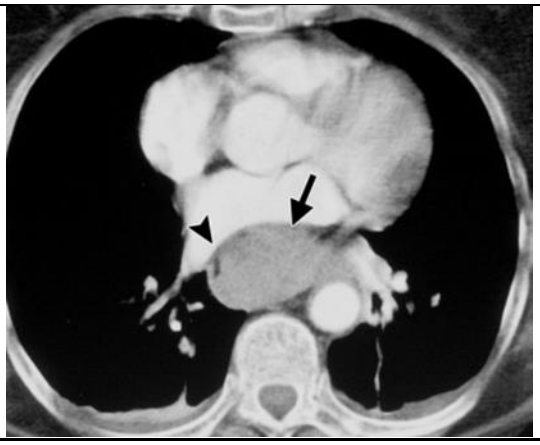
2) Mallory Weiss Tear

- Sudden increased intraluminal pressure due to:
  - Seizures – Child birth – Cough – Straining – Weight lifting
- Mucosal and submucosal tear without perforation



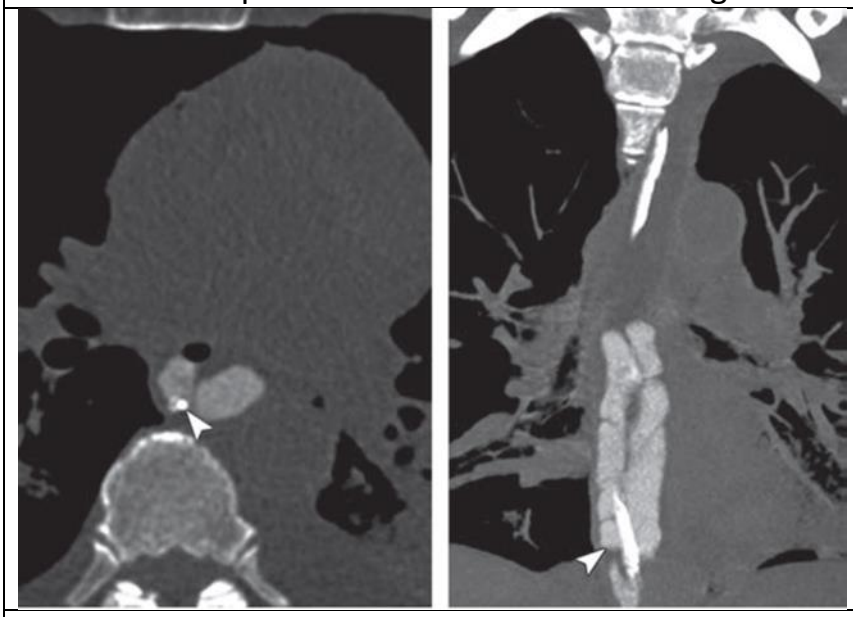


3) Esophageal Hematoma

Concentric Mural hematoma	Eccincentric Mural hematoma
	
	

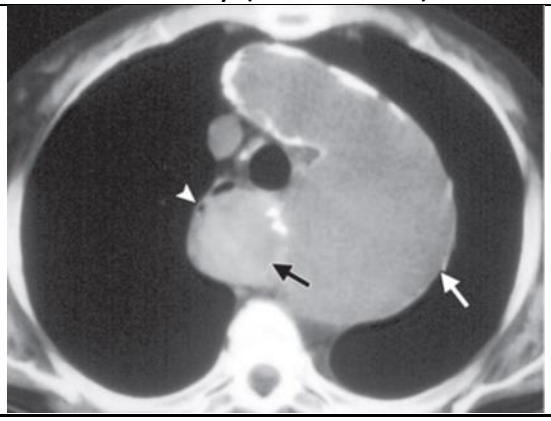
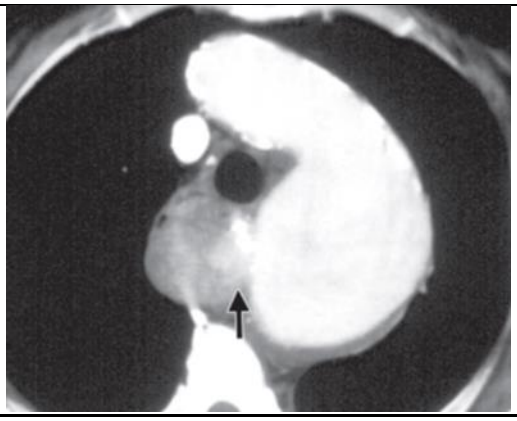
4) Esophageal Dissection

- History of recent instrumentation.
- Esophageal double-barreled appearance =
  Mucosal flap + Submucosal distribution of gas or oral contrast material



5) Aorto-Esophageal Fistula

- Aneurysmal dilatation of the thoracic aorta, which ruptures into the esophageal wall and leads to development of an intramural hematoma.
- The intramural hematoma displaces the esophageal lumen laterally (arrow).

Hyperattenuating (66-HU) esophageal hematoma (black arrow) displacing the esophageal lumen laterally (arrowhead)	Fistulous tract from a penetrating aortic ulcer (arrow)
	

6) Tracheo-Esophageal Fistula

Fistulous communication may develop between the esophagus and the trachea, bronchi, or lung parenchyma.

Benign causes:

- Corrosive esophageal injuries,
- Prolonged intubation,
- Surgical or endoscopic procedures,
- Esophageal infection,
- Simultaneous rupture of mediastinal lymph nodes into the esophagus and airways,
- Ingestion of foreign bodies

Malignant causes:

- Esophageal carcinoma,
- Bronchogenic carcinoma
- Lymphoma

Ba swallow: Oral contrast extravasation from esophagus into left bronchus.	Esophagotracheal fistula secondary to a swallowed inferior partial denture

Post Pulmonary Resection Complication

Pulmonary Resection Techniques:

- ❖ Pneumonectomy (Intrapleural, extrapleural, intrapericardial, and sleeve pneumonectomy)
- ❖ Lobectomy
- ❖ Limited resection (sleeve lobectomy, segmentectomy, nonanatomic parenchyma-sparing resection)

A) Pleural Space Complication:

- Hemothorax
- Chylothorax
- Empyema
- BPF

B) Lung Complication:

- Atelectasis
- Pneumonia
- Pulmonary edema
- Lung torsion

C) Airway Complication:

- Bronchial Anastomosis Dehiscence
- Bronchial Anastomosis Stenosis

Post pneumonectomy Space:

- ➢ First 24hrs => Filled with air
- ➢ In 1 week => 2/3 of the hemithorax filled with serosanguinous fluid
- ➢ In 2 weeks => 90% of the hemithorax is obliterated with fluid
- ➢ 4 months => Complete obliteration of the hemithorax with fluid

Successive films shows:

- ➢ Gradual fill in with fluid (by rate 2 rib space / day)
- ➢ Gradual decrease of air
- ➢ Gradual ipsilateral mediastinal shift

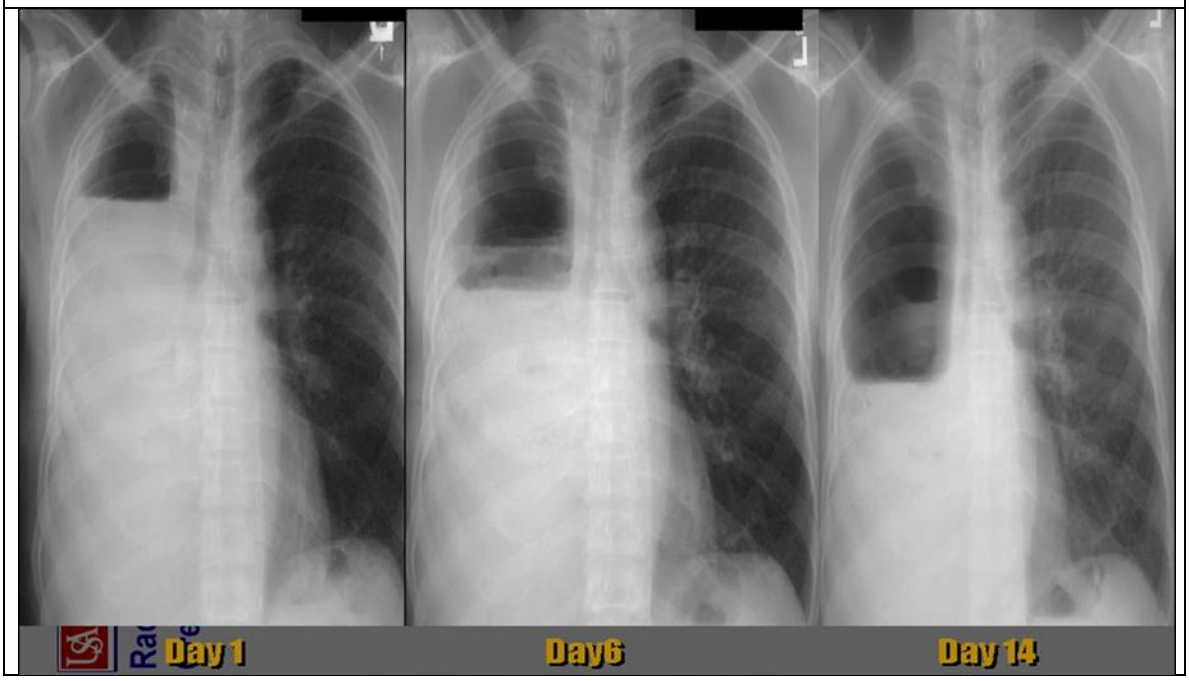
❖ Bronchopleural Fistula (BPF)

- 2-4% post pneumonectomy
- A fistula between bronchus and pleural space
- RT pneumonectomy > LT pneumonectomy
- BPF is the main cause of post pneumonectomy empyema

- **<u>Predisposing factors:</u>**
  - Active inflammation (TB)
  - Tumor in bronchial margins
  - Devascularized bronchial stump (Poor vascular supply)
  - Preoperative irradiation
  - Contamination of the pleural space

<u>Radiological findings:</u>

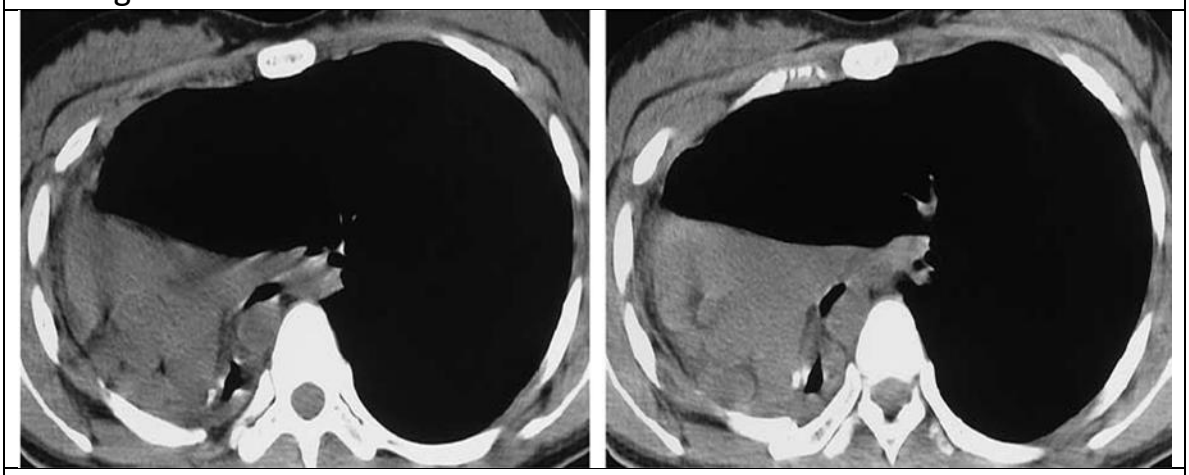
- Failure of progressive fluid filling in of the hemithorax
- Failure of ipsilateral mediastinal shift
- Persistent or progressive pneumothorax
- Gradual drop of the air / fluid level
- Re-appearance of the air / fluid level
- Contralateral mediastinal shift



❖ Post Pneumonectomy Syndrome

- A rare syndrome
- Rt pneumanectomy > Lt Pneumanectomy

- Marked mediastinal shift => Hilar structures rotation => Airway obstruction => Air trapping => Hyperinflated lung => Herniation of the lung into the contralateral side of the chest.



❖ Pneumonia

Postoperative pneumonia is most often caused by:

- ➢ Aspiration of gastric secretions
- ➢ Bacterial colonization of the atelectatic lobe
- ➢ Intubation and mechanical ventilation

<u>Radiological Findings:</u>

→ Patchy bronchopneumonic pattern: More common

→ Lobar airspace consolidation pattern: Less common

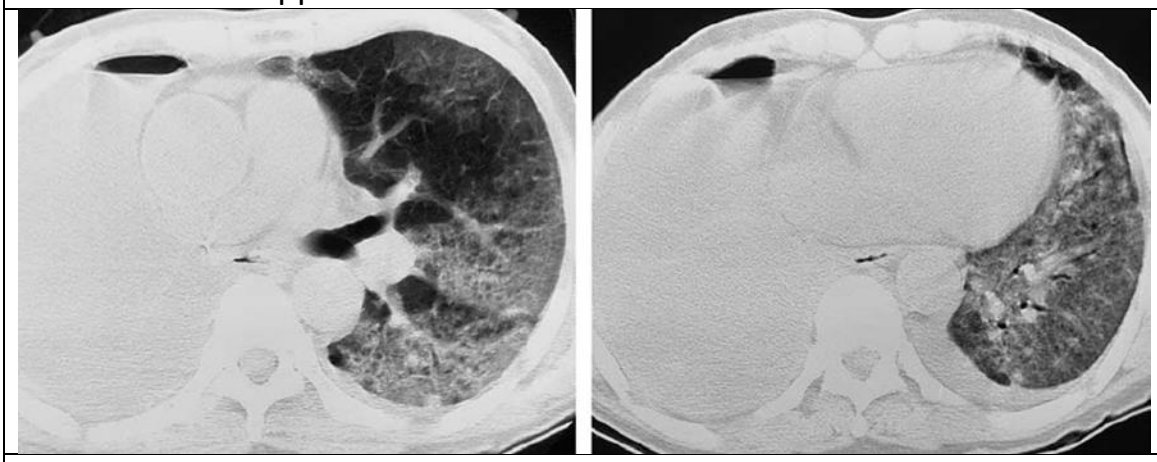
❖ Pulmonary Edema

- Postoperative pulmonary edema is a life-threatening complication that develops 2–3 days after pulmonary resection

Radiological Findings:

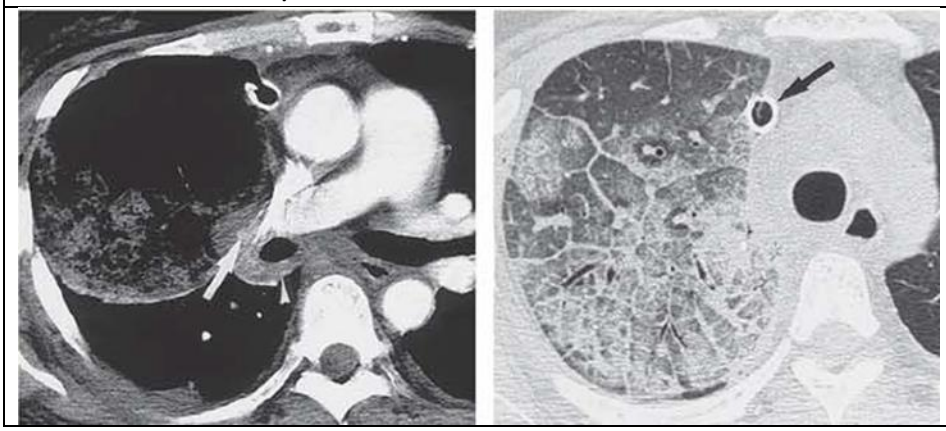
a) Milder form of postpneumonectomy pulmonary edema
   - Kerley lines, peribronchial cuffing
   - These findings have a tendency to disappear within a few days.

b) severe postpneumonectomy pulmonary edema:
   - Extensive ground glass opacities
   - An appearance identical to that of ARDS



❖ Lung Torsion

- Torsion of hilar structures and especially of the pulmonary veins => impaired venous flow => Interstitial edema and alveolar exudation.
- Complication: Hemorrhagic infarction – Ischemia – Necrosis.
- Most common: RT Middle lobe
- Consolidation / GGO + Volume increase



❖ Bronchial Anastomotic Dehiscence

- Extended lymph node dissection for lung cancer increases the risk of bronchial wall ischemia and anastomotic dehiscence.

CT Findings:

- Defect in the bronchial wall.
- Extraluminal air surrounding the anastomosis
- May lead to BPF

❖ Bronchial Anastomotic Stricture

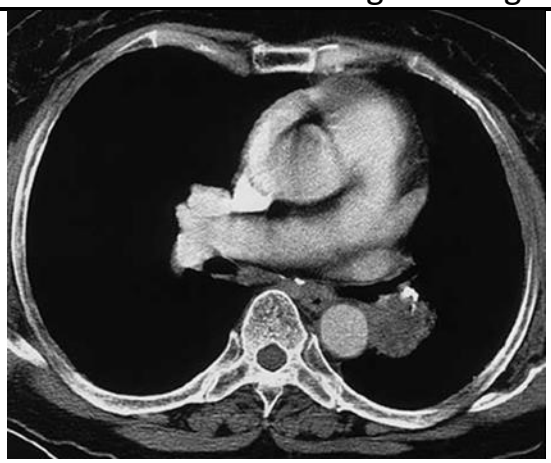
- The most common late complication
- CT: Bronchial narrowing at the anastomotic site.

❖ Cardiac Herniation with Torsion

- Very rare postpneumonectomy complication

❖ Local Tumor Recurrence

→ Mediastinal node metastasis
→ Recurrence in the chest wall or parietal pleura
→ Recurrence at the surgical margin



Post Lung Transplantation Complications

<u>Lung transplantation is an established treatment for Endstage pulmonary diseases:</u>

1) **Chronic Obstructive Pulmonary Disease (COPD) (39%)**
2) **Idiopathic Pulmonary Fibrosis (17%)**
3) **Cystic Fibrosis (16%)**
4) **Alpha-1-antitrypsin Deficiency (9%).**

Postoperative Complications of Lung Transplantation and Time of Occurrence

Time of Occurrence and Type of Complication
Immediate (<24 h)
Abnormality due to malposition of postoperative lines and tubes
Size mismatch between donor lung and recipient thoracic cage
Hyperacute rejection
Early (24 h to 1 wk)
Reperfusion injury
Pleural complications
Intermediate (8 d to 2 mo)
Acute rejection
Bronchial dehiscence
Pneumonia due to *Candida* infection
Primary late (2–4 mo)
CMV infection
Aspergillus infection
Pulmonary embolism
Bronchial stenosis
Native lung abnormality
Secondary late (≥4 mo)
Mycobacterial infection
Organizing pneumonia
Chronic rejection
Posttransplantation lymphoproliferative disorder
Primary disease recurrence
Upper-lobe fibrosis
Bronchogenic carcinoma

A- Malpositioned Monitoring Tubes and Lines (<24hrs)

- Accidental intubation of a main bronchus may result in rapid collapse of the contralateral lung
- Barotrauma from mechanical ventilation:
  - Pneumomediastinum,
  - Pneumothorax
  - Pulmonary interstitial emphysema.
- Improper placement of a central venous catheter or Swan-Ganz catheter leads to:

- Pneumothorax, Hemothorax
- Intra- and extrathoracic hematomas
- Perforation of the mediastinal vasculature
- Cardiac dysrhythmias

- Improper placement of a chest tube leads to:
  - Inadequate or ineffective drainage of postoperative air and fluid from the thoracic cavity.

B- Donor-Recipient Size Mismatch (<24 hrs)

- Size mismatch between donor lung and recipient thoracic cavity may cause mechanical complications

- If the donor lung is too large for the recipient thoracic cavity =>
  - Distortion of airways and atelectasis may occur, with retained secretions and secondary infections. This may lead to scarring.

- If the donor lung is too small for the recipient thoracic cavity =>
  - Graft hyperexpansion may lead to hemodynamic compromise,
  - Limited exercise tolerance, or frank pulmonary hypertension, all because of an inadequate vascular bed

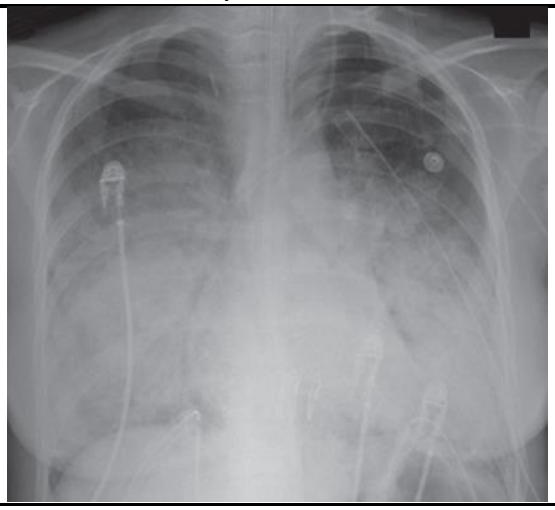
C- Ischemia-Reperfusion Injury (Reperfusion Edema = Reimplantation Response)

Ischemia-reperfusion injury is a noncardiogenic pulmonary edema

- Occurs more than 24 hours after transplantation
- Peaks in severity on postoperative day 4
- Improves by the end of the 1st week

Radiological Findings:

- Bilateral perihilar and basal air-space consolidation & GGO.



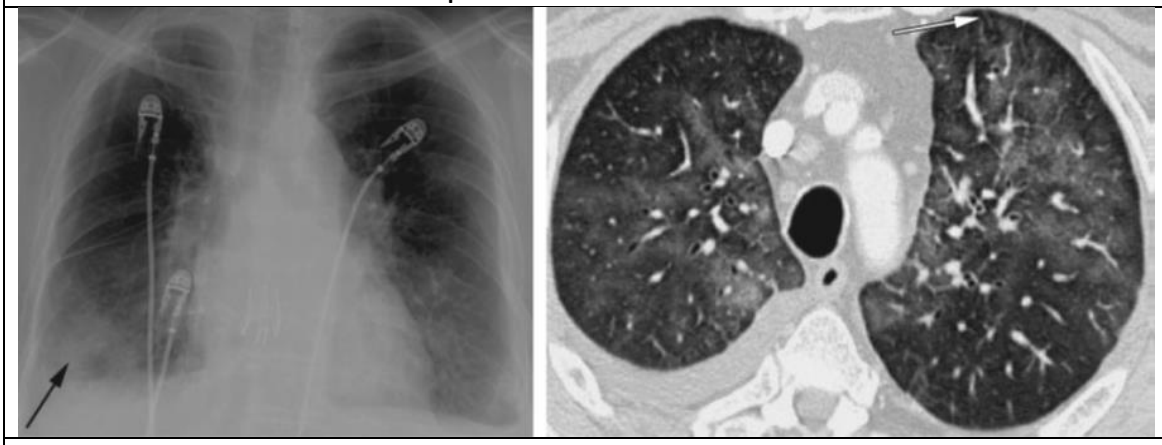
D- Rejection:

i) Hyperacute Rejection (<24 hrs)

- Fulminant, rapidly evolving, fatal clinical syndrome that may occur immediately after transplantation.
- Diffuse homogeneous infiltration of the entire allograft

ii) Acute Rejection (2w – 2m):

- Pattern:
  - Interstitial pattern: Peribronchial cuffing, inter- and intralobular septal thickening
  - GGO & alveolar edema
  - Pleural effusion
- Distribution: Bilateral perihilar and basal



iii) Chronic Rejection (Late Complication >4m):

- **Bronchiolitis obliterans:**
  - Centrilobular ground glass nodules
  - Tree-in-bud pattern
  - Mosaic attenuation
  - Air-trapping on expiratory CT
  - Bronchiectasis
  - Bronchial wall thickening

E- Airway Complications:

1) Bronchial dehiscence.
2) Bronchial stenosis
3) Bronchomalacia
4) Bronchovascular, bronchopleural, and bronchomediastinal fistula

1) Bronchial Dehiscence:

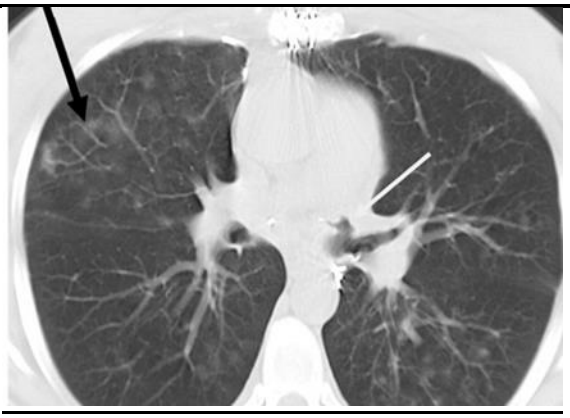
- is the most common airway complication
- in the early postoperative period, affecting 2–3% of cases
- Typically occurring 2–4 weeks after transplantation

Direct CT signs:

- Extraluminal gas – Focal bronchial wall defect

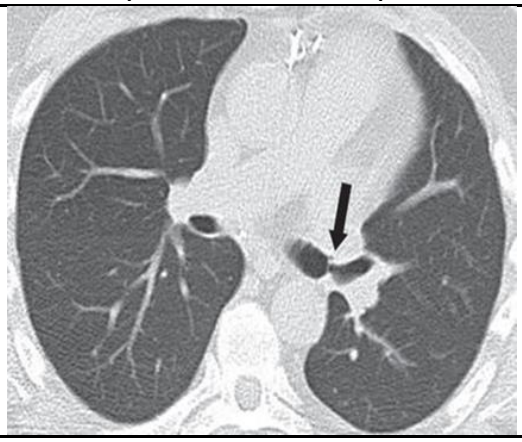
Indirect CT signs:

- New or persistent air leak, pneumothorax, and pneumomediastinum



2) Bronchial Stricture

CT is particularly useful in assessing the extent of bronchial stenoses in order to plan bronchoscopic stent insertion



F- Pleural Complications:

Pneumothorax – Pleural Effusion – Empyema

G- Infections:

- Most common complication after transplantation and is a major cause of morbidity and mortality
- Because of immunosuppression, lung denervation and loss of the cough reflex, impaired mucociliary function, and lymphatic drainage

- Bacterial infections: Pseudomonas aeruginosa and Klebsiella species
- Viral infections: CMV infection.
- Fungal infections: Candida and Aspergillus organisms
- Pneumocystis pneumonia is now uncommon because of the routine use of trimethoprimsulfamethoxazole prophylaxis

H- Cryptogenic Organizing Pneumonia

- Pattern: Air space consolidation & GGO
- Atoll sign: Central lucency surrounded by peripheral opacity.
- Distribution: Peripheral & Basal & Migratory

I- Pulmonary Embolism and Infarction

- Pulmonary thromboembolic events tend to occur within 4 months after transplantation, and most occur within the allograft

- **<u>Vascular findings of Acute PE:</u>**
  1) Complete filling defect + Enlargement of the occluded artery
  2) Partial central filling defect surrounded by contrast material enhancement = “railway track” sign on longitudinal images
  3) Partial peripheral filling defect + Acute angles with the arterial wall

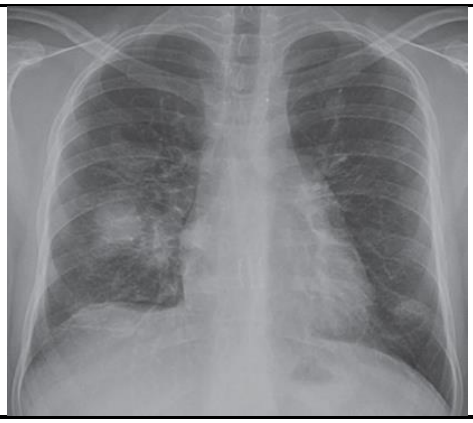
- **<u>Parenchymal findings:</u>**
  - Wedge-shaped consolidation,
  - Mosaic attenuation or oligemia
  - Atelectasis
  - Pleural effusion

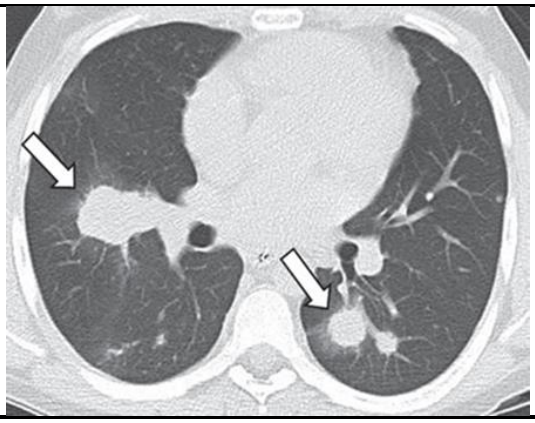
J- Post-Transplantation Lymphoproliferative Disorder

- PTLD is a spectrum of diseases that vary from a histologically benign polyclonal lymphoid proliferation to aggressive high-grade lymphoma
- Epstein-Barr virus is found in 90% of patients with this disorder
- It may manifest from 1 month to several years after transplantation but tends to occur within the first year, peaking at 3–4 months.

Radiologically findings:

- Solitary or multiple pulmonary nodules or masses
- Halo sign: nodules surrounded by GGO
- Hilar or mediastinal adenopathy
- Thymic enlargement
- Pleural effusion
- Pericardial masses is less common





K- Upper Lobe Fibrosis

Upper lobe fibrosis is uncommon
Reported to occur 18–72 months after lung transplantation

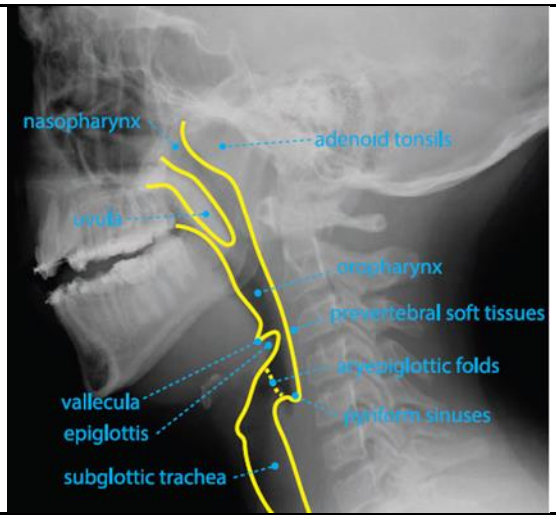
CT findings

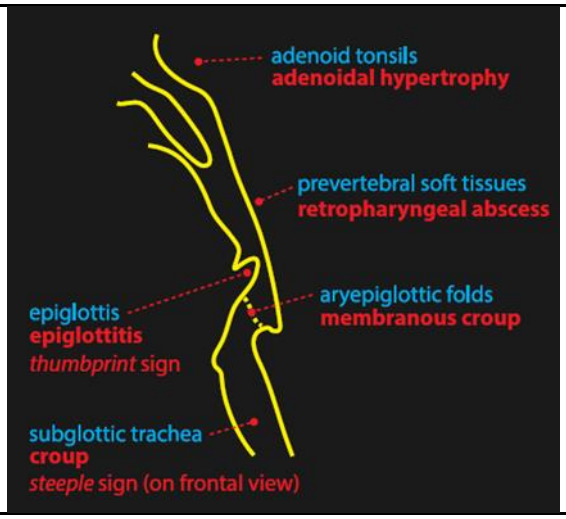
- Interlobular septal thickening
- Gradual development of coarse reticular opacities
- Traction bronchiectasis, honeycombing, architectural distortion, and volume loss.

L- Recurrence of Primary Disease

- Recurrence of primary disease in the allograft may appear as early as 2 weeks or as late as 2 years after transplantation.
- Sarcoidosis, lymphangioleiomyomatosis, bronchioloalveolar carcinoma, and Langerhans cell histiocytosis

Pediatric Stridor





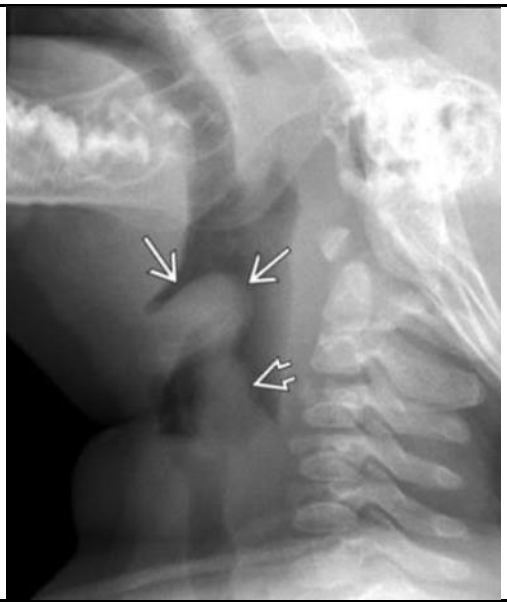
1) Epiglottitis

Medical emergency caused by infectious inflammation of the epiglottis

Most commonly caused by: Haemophilus Influenza
Age = 3-6y

Lateral X-ray:

- **Thickening epiglottis (Arrow) (Thumbprint sign)**
- **Thickened aryepiglottic folds (Open arrow)**



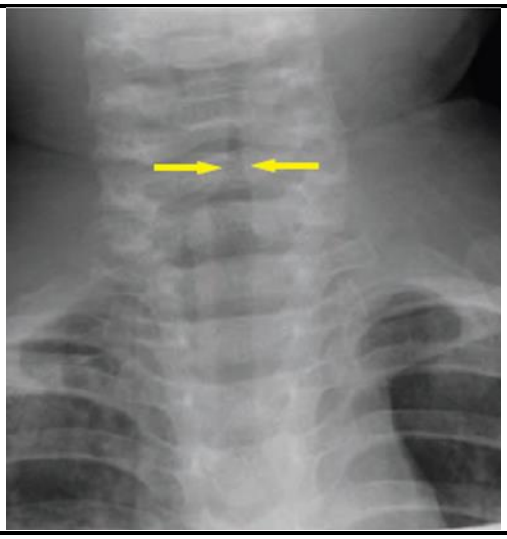
2) Croup (Laryngotracheobronchitis)

Croup is a viral (usually parainfluenza) infection with characteristic inspiratory stridor and barking cough.

Age = 6m – 3y

X-ray AP view:

Subglottic narrowing
Inverted V
Steeple sign = loss of the normal shouldering of the subglottic trachea.



3) Aspirated Foreign Body

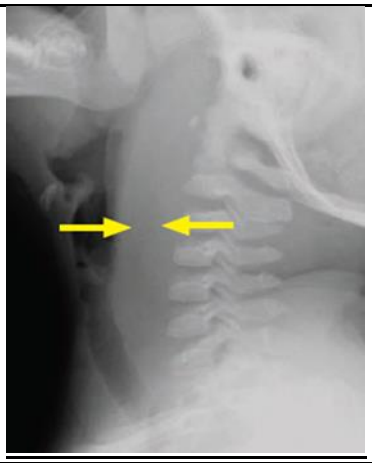
- Radiography can't directly visualize a radiolucent FB
- Secondary signs of air trapping can suggest the diagnosis.

4) Retropharyngeal Abscess

- A purulent infection of the retropharyngeal space
- One of the more common causes of nontraumatic prevertebral soft tissue swelling in children.

X-ray Latera view:

- Widening of the retropharyngeal space
- DD:
  - Retropharyngeal hematoma
  - Lymphoma
  - RMS



5) Exudative (Bacterial) Tracheitis

- Clinically present similarly to epiglottitis with fever, stridor, and respiratory distress
- is also potentially life-threatening.
- In contrast to croup, exudative tracheitis tends to affect older children
- Causative organism: Bacterial (most commonly S. aureus).
- On imaging, intraluminal membranes may be visible in the subglottic and cervical trachea.

6) Subglottic Hemangioma

- A benign vascular neoplasm that produces stridor in infancy.
- It is the most common pediatric subglottic tracheal mass.
- Subglottic hemangioma is often associated with cutaneous hemangiomata.
- The classic radiographic finding is asymmetric narrowing of the subglottic trachea on the frontal view.

7) Laryngeal Papillomatosis:

- Laryngeal papillomatosis is a cause of multiple laryngeal nodules due to HPV infection, resulting in thick and nodular vocal cords.
- Papillomatosis increases the risk of laryngeal squamous cell carcinoma (analogous to cervical cancer risk from HPV).
- Papillomas may rarely seed the lungs, causing multiple cavitary nodules.

8) Laryngomalacia

- Common cause of stridor in the 1st year of life.
- Immature laryngeal cartilage
- Laryngeal supraglottic collapse with inspiration
- Relieved by prone position or neck extension.

9) Tracheomalacia

- Collapse of trachea with expiration
- Focal or diffuse
- Focal type: usually secondary to congenital vascular ring

Congenital High Airway Obstruction Syndrome (CHOAS)

1) Tracheal / Laryngeal Atresia

2) Tracheal / Laryngeal Web

3) Congenital Tracheal Stenosis

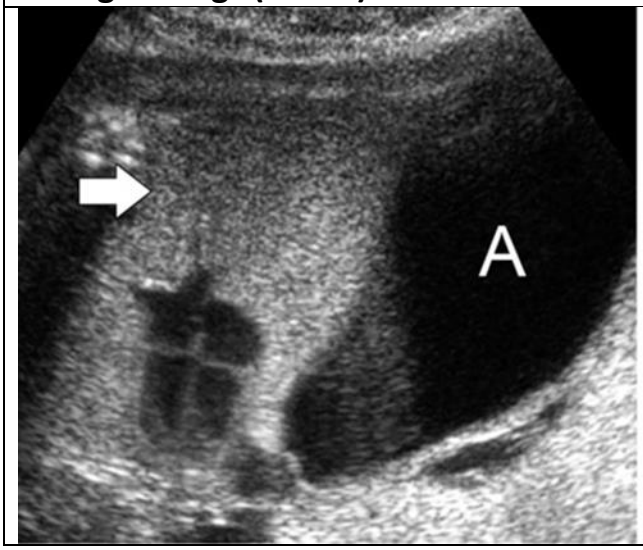
→ Generalized stenosis 30%
→ Focal stenosis (Ring like) 50%
→ Carrot- or funnel-shaped segmental stenosis 20%

4) Subglottic Stenosis

→ Fixed narrowing at the level of cricoid
→ Due to failure of laryngeal recanalization in utero.

Fetal US / MRI:

- Symmetrically enlarged echogenic lungs / high T2 signal
- Dilated and fluid-filled trachea and bronchi
- Inverted hemidiaphragms
- The heart
    - Appears small compared with the enlarged lungs
    - Appears anteriorly positioned due to compression by lungs.
- Massive ascites.
- Hydrops may be present.

Enlarged and echogenic lungs	Flat diaphragm
Enlarged lungs (arrow) + Massive ascites (A).	
	

Bronchopulmonary Foregut Malformations

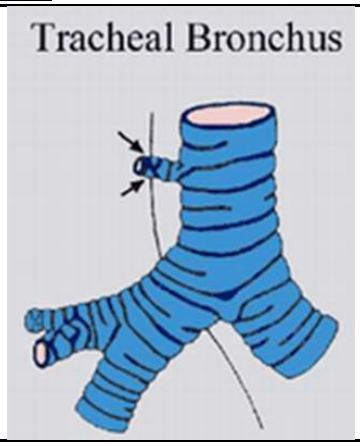
❖ Congenital Bronchial Abnormalities

1) Tracheal Bronchus

- Right upper bronchus originating in the trachea
- Exits the right lateral wall of the trachea < 2 cm above the major carina
- can supply the entire upper lobe or its apical segment

Replaced = Anatomic upper-lobe bronchus is missing
Supernumerary= if the right upper-lobe bronchus has a normal trifurcation into apical, posterior, and anterior segmental bronchi
Tracheal Diverticulum :- supernumerary bronchi may end blindly

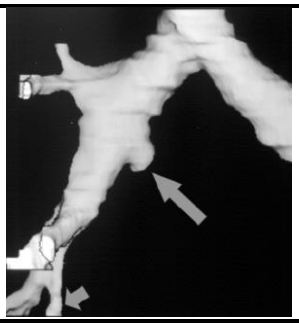
Right tracheal bronchus = 0.1%–2%
Left tracheal bronchus = 0.3%–1%



2) Accessory Cardiac Bronchus

- Arises from inferior medial wall of the right main or intermediate bronchus
- It may end blindly or be associated with small amounts of abnormal pulmonary parenchyma.

- Complication: Bronchocele – Bronchiectasis – Hemorrhage – Infection.



❖ Congenital Pulmonary Airway Malformation (CPAM): = Congenital Cystic Adenomatoid Malformation (CCAM):

- Heterogeneous group of cystic and noncystic lung lesions that largely result from early airway maldevelopment.
- Cysts communicate with bronchial tree at birth and fill with air early in life

Classification:

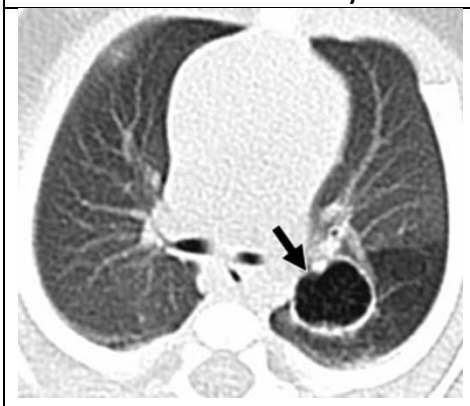
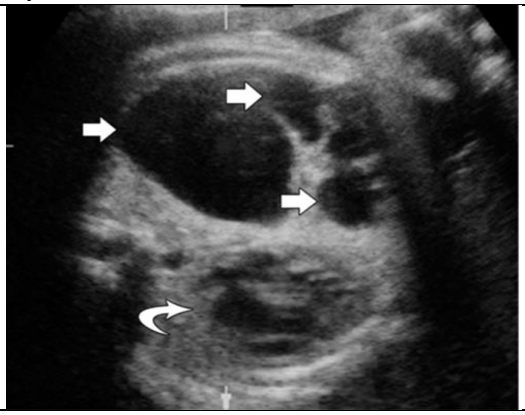
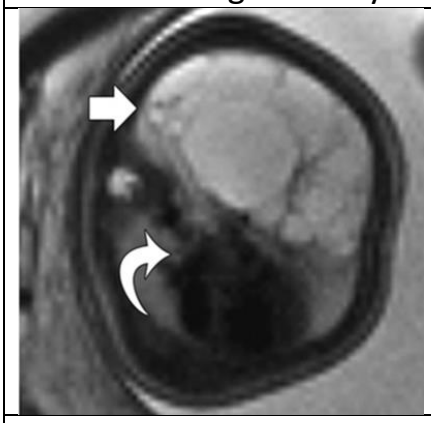
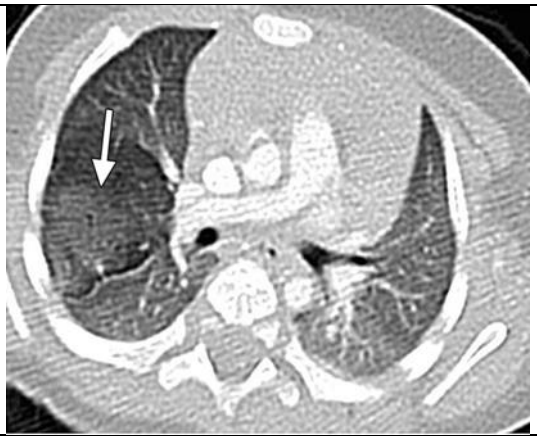
Type 0 has a tracheal or bronchial origin and is really acinar dysgenesis or dysplasia,
Type 1 has a bronchial or bronchiolar origin (large [2–10-cm] cyst lesion)
Type 2 has a bronchiolar origin (small [0.5–2-cm] cyst lesion)
Type 3 has a bronchiolar–alveolar duct origin (adenomatoid type)
Type 4 has a distal acinar origin (the "unlined" cyst lesion).

3 Radiological patterns described by Stocker et al:

- **CCAM type I (50%):**
  - 1 or more large cysts (2-10 cm)
- **CCAM type II (40%):**
  - Numerous small cysts (0.5-2 cm)
- **CCAM type III (< 10%):**
  - Appears solid on gross inspection and imaging but have microcysts (< 0.5 cm)

Location:

- No lobar predilection
- Most lesions confined to single lobe
- Most lesions solitary

CPAM / CCAM Type I	
Postnatal CT: Solitary well-defined thin walled air-filled cyst	Fetal US: Multiple large anechoic cysts
	
Fetal MRI: Large multicystic lesion with high signal intensity	
	
CPAM / CCAM type II	**CPAM / CCAM type III**
CT: Multiple small cystic lesions	CT: Ill-defined area of increased attenuation
	

❖ Bronchogenic Cyst

Foregut duplication cysts consisting of:

- Bronchogenic cyst
- Enteric cyst
- Neuroenteric cyst.

Location:

Middle Mediastinum (65%–90%):

→ Subcarinal location is most common.

→ Right paratracheal

→ Hilar regions.

Pulmonary:

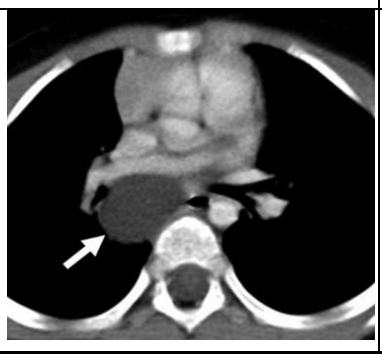
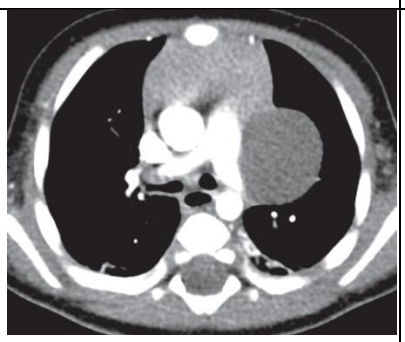
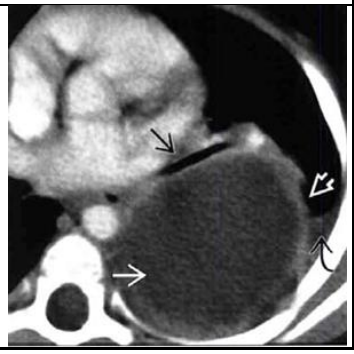
→ Majority in the medial third of the lungs

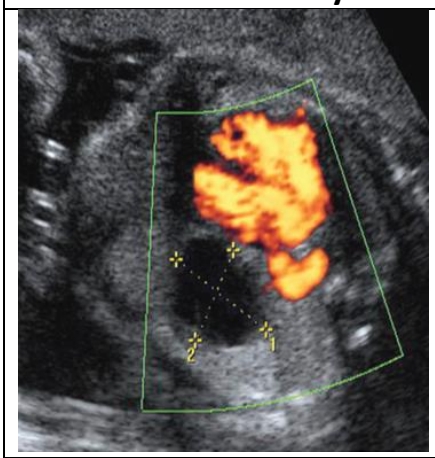
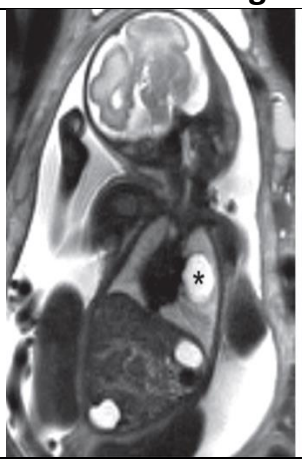
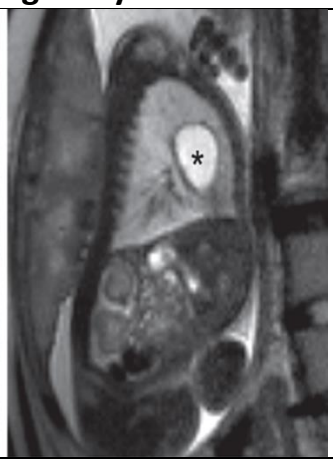
→ More frequent in the lower lobes

→ Equal incidence in both lungs

Rare: in thymus, diaphragm, neck, pericardium, and retroperitoneum

CT: Well defined thin walled fluid attenuation cyst (0-20 HU)

Subcarinal	Hilar	Pulmonary
		

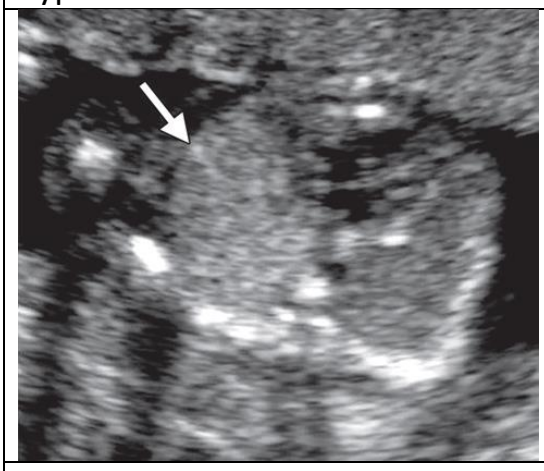
Fetal US: Anechoic cyst	Fetal MRI: T2 high signal cyst	
		

❖ Congenital Lobar Emphysema

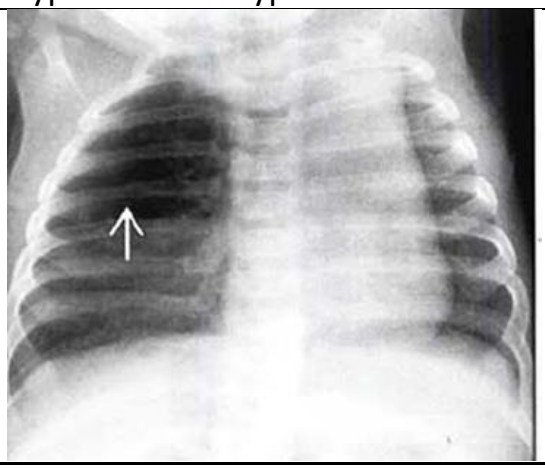
- Characterized by progressive lobar overexpansion, usually with compression of the remaining (ipsilateral) lung

Location:

→ Left upper lobe in 42.2%
→ Right middle lobe in 35.3%
→ Right upper lobe in 20.7%
→ Either lower lobe in < 1%

Fetal US: Large homogenous hyperechoic mass	Fetal MRI: high T2 signal
	

Hyperlucent + Hyperinflated hemithorax => Mediastinal shift



❖ Bronchial Atresia

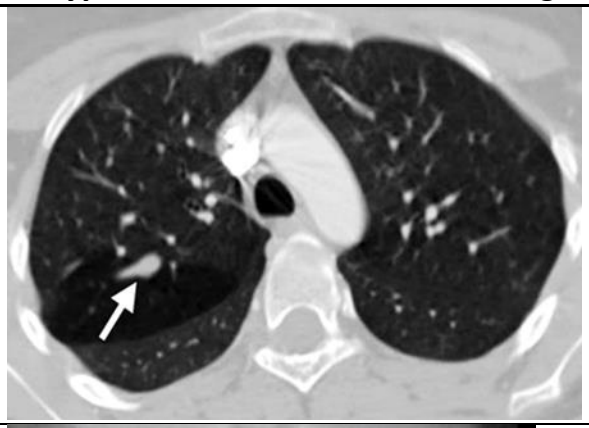
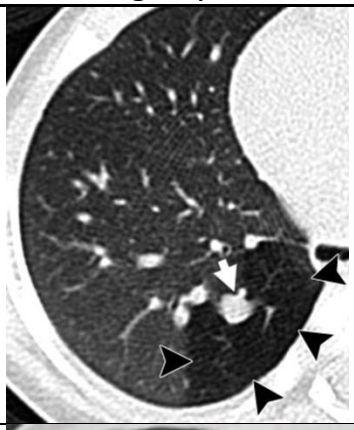
- Rare anomaly resulting from focal obliteration of a segmental, subsegmental, or lobar bronchus.
- The bronchi distal to the stenosis are dilated and filled with mucus
- Mild hyperinflation of the adjacent lung due to collateral air drift via the interstitium and pores of Kohn

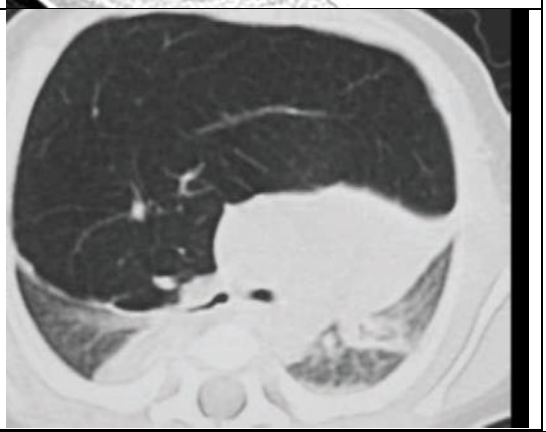
Location:

→ Left upper lobe (Most common location).
→ Right upper lobe
→ Right middle lobe
→ Lower lobe bronchi rare

CT:

- **Finger in glove:** Central mass like opacity adjacent to the hilum that may have a tubular configuration representing the obstructed, dilated, and mucoid-filled segmental or subsegmental bronchus.
- **Hyperinflation** in the involved segment of lung beyond the obstruction.



❖ Bronchopulmonary Sequestration (PBS)

- 2nd most common lung lesion (after CPAM) detected antenatally .

Characterized by:

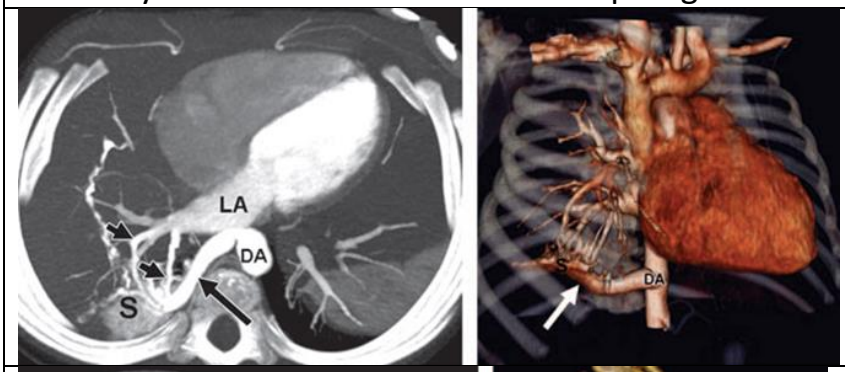
- ➢ Dysplastic and nonfunctioning pulmonary tissue
- ➢ NO connection with the tracheobronchial tree
- ➢ Its blood supply comes from systemic vessels.

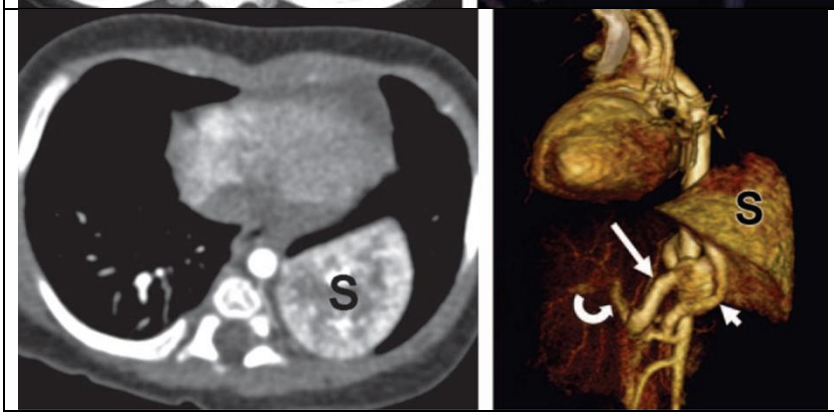
Extralobar BPS	Intralobar BPS
Less common (25% of cases)	More common (75% of cases)
Diagnosed in the prenatal-neonatal period.	Diagnosed later in childhood or adulthood.
Has its own pleural investment	Within visceral pleura
Systemic venous drainage : Azygous vein and less commonly into the portal vein, left subclavian vein, or the internal mammary vein	Pulmonary venous drainage: Inferior pulmonary vein
Systemic arterial supply from aorta	

Location:

98% in the lower lobes LT > RT

20% may arise from below the hemidiaphragm





Pulmonary Venolobar Syndrome / Hypogenetic Lung Syndrome Scimitar Syndrome

- PAPVR.
- Pulmonary hypoplasia
- Dextroposition of the heart
- Anomalous systemic arterial supply to the right lung

❖ Congenital Diaphragmatic Hernia

CDH is caused by a defect in the diaphragmatic musculature, with herniation of the abdominal viscera into the thorax leading to deficiencies in lung development

- Ipsilateral lung: Hypoplastic pulmonary changes
- Contralateral lung: affected by mediastinal shift and compression

Location:

Bochdalek hernia: Posterior hernia, most common type of CDH

Morgagni hernia: Anterior hernia, much less common in Neonates

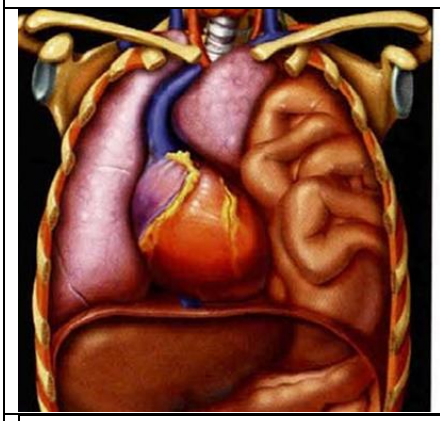
92% of CDHs are posterior lateral defects:

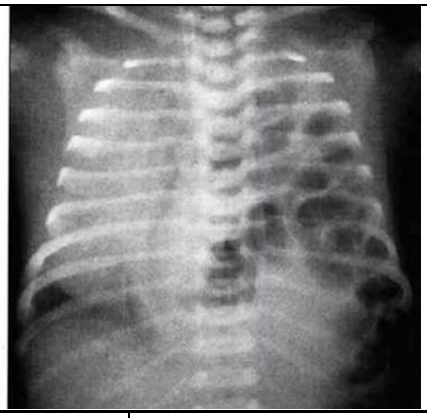
97% Unilateral

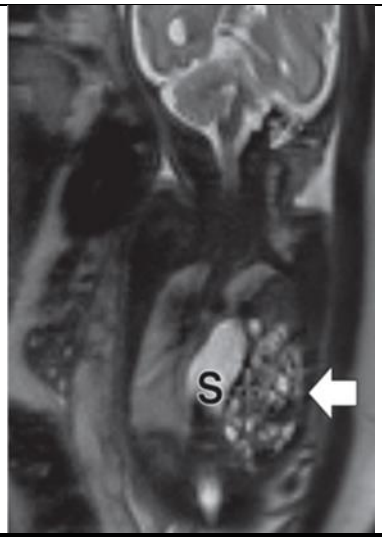
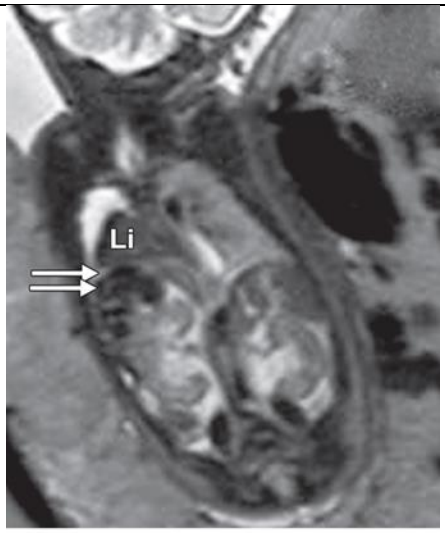
80%-90% Left side => Herniation of fluid filled stomach and bowel

10% Right side => Herniation of liver

5% Bilateral





LT side CDH	RT side CDH
	

❖ Congenital Hydrothorax

Uni- and bilateral pleural fluid collections, which are considered abnormal in any gestational phase

Fetal intervention (thoracentesis or thoracoamniotic shunting) is reserved for hydrothorax with a marked mass effect leading to a mediastinal shift.

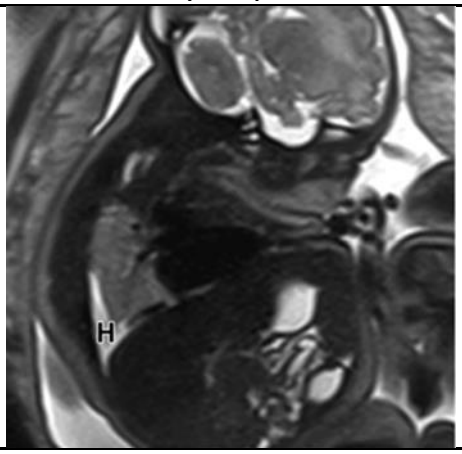
Primary cause:

- Chylothorax: chylous leak caused by lymphatic duct abnormality that result in defective chest drainage.

Secondary causes:

- Generalized fluid retention related to fetal hydrops

Fetal MRI: fluid collection surrounding the lungs that is hypointense on T1 and hyperintense on T2



❖ Congenital Pulmonary Lymphangiectasia

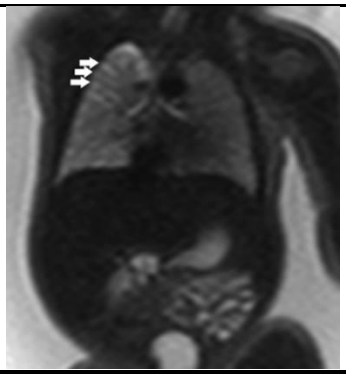
is a generalized dilatation of the lymphatic vessels, including pulmonary, subpleural, interlobar, perivascular, and peribronchial dilatation

A) Congenital form is usually fatal in early life, and it probably results from pulmonary interstitial connective tissue failure, leading to dilatation of the pulmonary lymphatic vessels.

B) Secondary lymphangiectasia may be due to surgery, infection, tumor genesis, or, in cases of total anomalous pulmonary venous return or hypoplastic left heart syndrome, venous obstruction

Fetal MRI: T2 WI:

- Bilateral inhomogeneity of the lung parenchyma.
- High signal intensity is seen in the pulmonary interstitium very often associated with pleural effusion



Neonatal Respiratory Distress

4 main diseases:

1) Respiratory Distress Syndrome (RDS)/Hyaline Membrane Disease (HMD)

2) Transient Tachypnea Of The Newborn (TTN)

3) Meconium Aspiration

4) Neonatal Pneumonia

Most common complications:

a. **Pulmonary interstitial emphysema (PIE)**
b. **Bronchopulmonary dysplasia (BPD)**
c. **Persistent PDA**

1) Respiratory Distress Syndrome (RDS) / Hyaline Membrane Disease (HMD)

Aetiology: ↓ Surfactant => Alveolar atelectasis

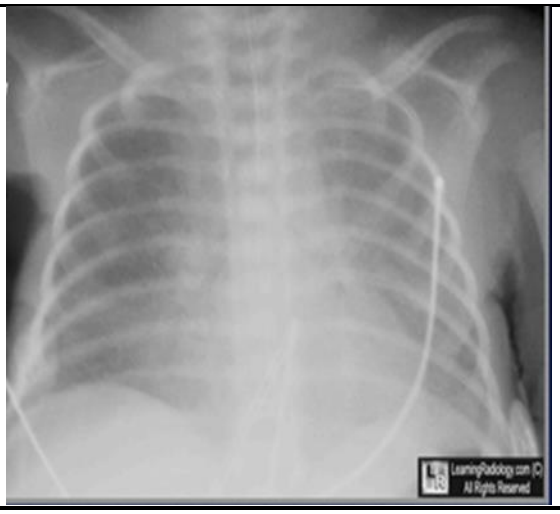
Predisposing factors:

- Premature Infants - Cesarean section – Infants of diabetic mother

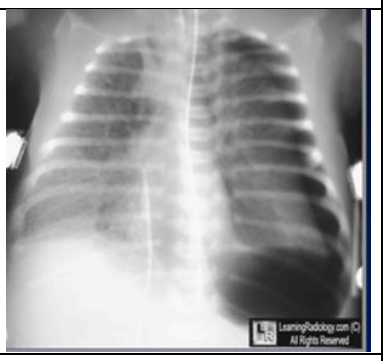
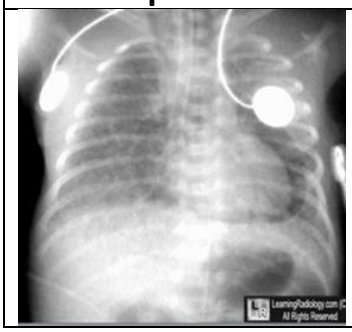
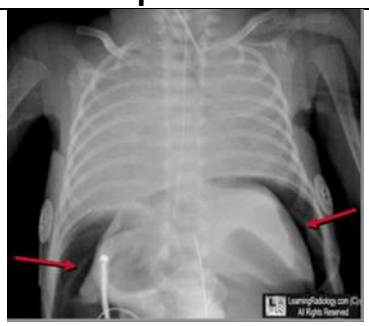
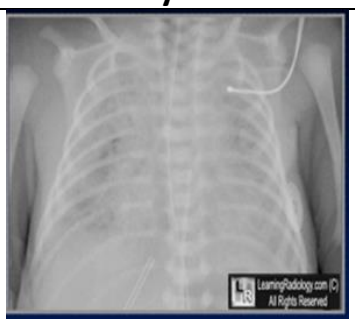
Age of presentation: First 2hrs of life

CXR:

- Decreased lung volume
- Bell shaped thorax
- Bilateral & symmetrical
- Ground glass opacities
- Granularity (Alveologram)
- Air bronchogram



Complications:

Pulmonary interstitial emphysema (PIE)	Pneumomediastinum	Pneumothorax
		
Pneumopericardium	**Pneumoperitoneum**	**Pulmonary edema**
		

2) Pulmonary Interstitial Edema (PIE):

- Is a complication of positive pressure ventilation (Barotrauma).
- Accumulation of air into peribronchial and perivascular (interstitial) spaces and spread along lymphatics.
- CXR: Tortuous lineal lucencies radiate outward from hilar region.

3) Bronchopulmonary Dysplasia (BPD) / Chronic Lung Disease of Prematurity

- BPD is a consequence of early acute lung disease.
  - Complicate HMD
  - Also meconium aspiration syndrome and pneumonia
- BPD is clinically defined as abnormal chest radiograph and persistent need for oxygen at 36 post-conceptual weeks or at 28 days of life.
- BPD is the most common cause of chronic respiratory failure in pediatrics.

Stages of BPD:

- Stage I (0-3days): Ground glass opacities + Air bronchogram (as HMD)
- Stage II (4-10days): Complete opacification + white lung + air bronchogram
- Stage III (10-20days): Small round cystic lucencies (honeycombing).
- Stage IV (>1month): Hyperiflation + linear atelectasis + Emphysema (as meconium aspiration)

4) Meconium Aspiration:

Fetal distress => In utero defecation => Aspiration of meconium stained amniotic fluid

- ⇨ Chemical irritant => Chemical pneumonitis
  - ⇨ Bilateral patchy opacities
- ⇨ Ball and valve airway obstruction
  - ⇨ Ropey densities = Emphysema + Air trapping + Atelectasis
  - ⇨ Hyperinflated lungs
  - ⇨ Pneumothorax – Pneumomediastinum (25%)

5) Transient Tachypnea of the Newborn (TTN) = Wet Lung Syndrome:

- A clinical diagnosis caused by delayed resorption of intrauterine pulmonary liquids.
- Caused by:
  - Cesarean section – Premature delivery
  - Maternal sedation (No thoracic squeezing)
  - Hypoproteinemia – Hypervolemia – Erythrocythemia.

- Age of patient: Full term infant
- Presenting immediately after birth
- Clear in 24 – 48hrs

CXR: Fluid overload = Non-cardiogenic pulmonary edema:

- Prominent vascular markings
- Pleural effusion - Fluid in fissure - Alveolar edema

6) Neonatal Pneumonia

- Pathogenesis:
  - Transplacental infection: TORCH
  - Perineal infection: Group B Streptococcus, S. aureus, and E. coli

CXR: patchy asymmetrical opacities

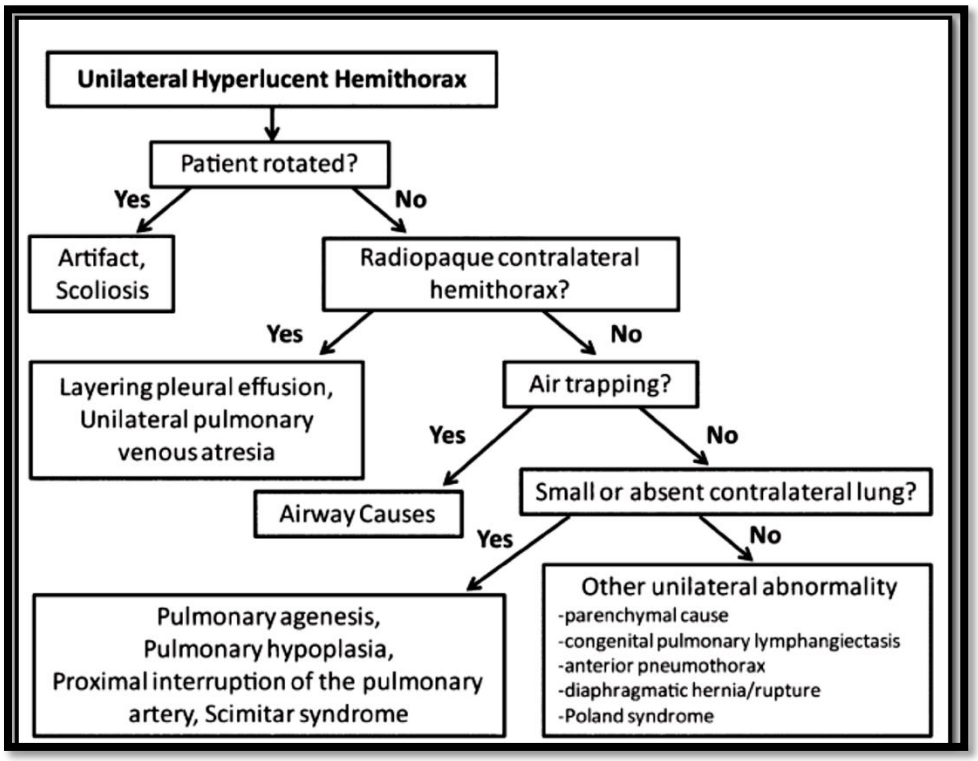
Unilateral Hyperlucent Hemithorax

- **Rotation**
  - Poor patient positioning
  - Scoliosis
- **Chest wall defect**
  - Mastectomy
  - Poland syndrome (absent pectoralis muscle)

- **Pleural**
  - Pneumothorax
  - Pleural effusion (contralateral)
- **Mediastinal**
  - Mediastinal fibrosis

- **Pulmonary (ventilation)**
  - Airway obstruction
  - Bronchial compression (hilar mass, cardiomegaly)
  - Endobronchial obstruction with air trapping (foreign body, tumour, mucous plug)
  - Obliterative bronchiolitis
  - Swyer-james syndrome
  - Pulmonary emphysema (asymmetric)
  - Congenital lobar emphysema
  - Unilateral bullus / bullae
  - Compensatory hyperinflation
  - Pneumonectomy

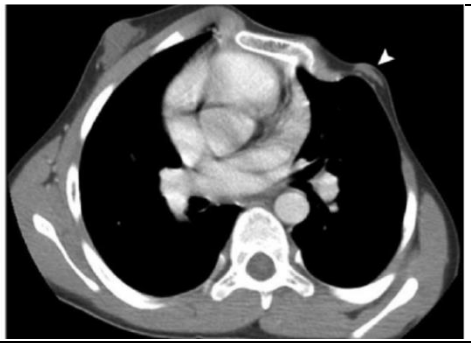
- **Vascular (Perfusion)**
  - Pulmonary agenesis / hypoplasia
  - Pulmonary embolism
  - Congenital heart disease
  - Congenital pulmonary artery hypoplasia / stenosis
  - Shunt (e.G. Blalock taussig)
  - Unilateral absence pulmonary artery (UAPA)

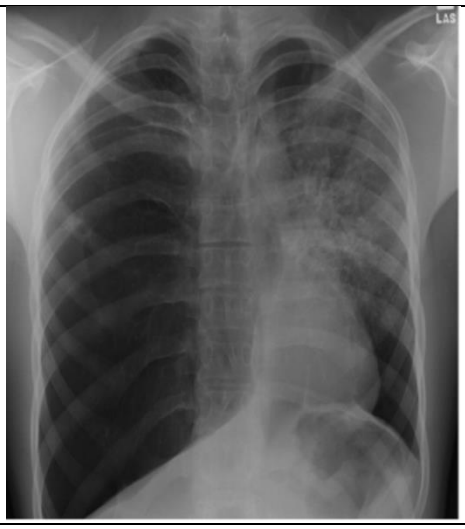
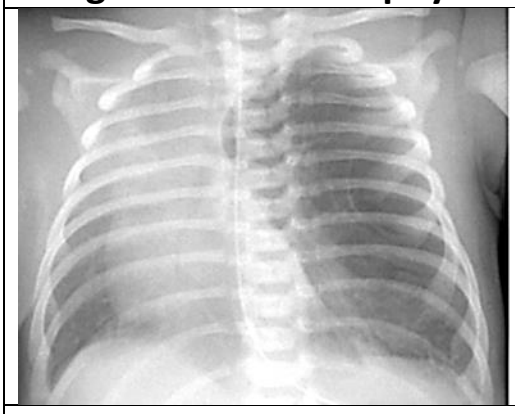
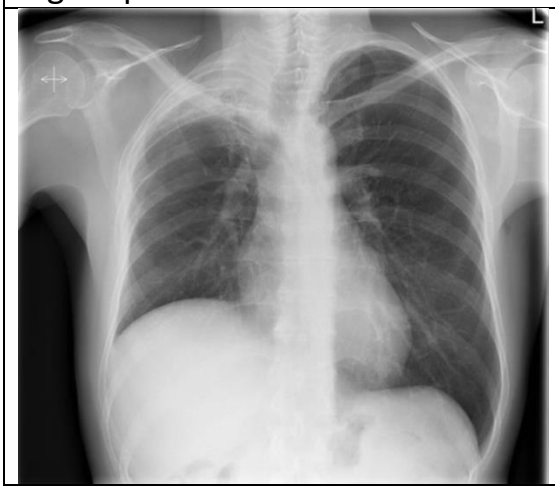


Rotation

Mastectomy

Poland Syndrome



Pneumothorax	**Pneumatocele**
	
Congenital Lobar Emphysema	
	
Compensatory Emphysema Right apical fibrosis	**MacLeod Syndrome**